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THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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WASTING AND RECOVERY OF ZOSTERA MARINA ON THE ATLANTIC COAST OF THE UNITED STATES[1]

Neil E. Stevens, Hazel R. Ellis, and Russell B. Stevens

Measured by its effect on the host plant, the wasting disease of Zostera marina is the most devastating in the history of plant pathology. In a period certainly not longer than two seasons this native plant was all but exterminated throughout its range on the Atlantic coast of North America and much of Europe. In discussing this apparently unique plant disease we are justified, like paleobotanists, in picking up and examining the smallest bits of evidence. Epidemics of the wasting disease do not come every few years and the North Atlantic is not convenient for replicated field plots.

Clarence Cottam, Assistant Director of the Fish and Wildlife Service, was one of the first workers in the United States to take an interest in this disease. He is our authority for the statement that no place is known to coastal wardens or other workers in which the disease was not serious. This contrasts, of course, with the condition reported in the southern Baltic.

The wasting disease struck the Atlantic coast with greatest force in 1931. In some areas, Upper Buzzards Bay, Massachusetts, for example, most of the destruction occurred in 1932. Certainly throughout the area the normally dense beds had been nearly or quite destroyed by 1933.

What portion of the plants did survive in the United States? Of course no one can ever say accurately. Stevens ventured an estimate that the plants surviving the epidemic in Upper Buzzards Bay comprised less than one hundredth of one percent of the original stand. In Buttermilk Bay less than ten square yards of Zostera marina could be found in 1933 or 1934 in an area which in 1930 certainly had more than 750,000 square yards of this species. Although eelgrass was not exterminated, it came closer to extermination than is generally recognized, closer perhaps than any plant of which we have record, with the possible exception of the American chestnut. Individual plants which survived such a slaughter either had some resistance to the disease or were extremely "fortunate".

What Caused the Wasting Disease?

It is now generally recognized that three interacting factors are involved in the biological phenomenon we call pathogenic disease: a host plant, an adapted pathogen, and a favorable environment. Marked change in the severity of a given disease may be caused by marked change in any one of these three elements.

It is inconceivable that a native seed plant should have undergone such a radical change so suddenly over some thousands of miles of coast on both sides of the Atlantic Ocean. There is general agreement, on the American side of the Atlantic at least, that the proximate cause of the wasting disease was parasitism by a Labyrinthula[2] still widely present on eelgrass in some localities. It is difficult, if not impossible, to imagine this organism to have mutated suddenly on both sides of the Atlantic. Many, perhaps most, of those still actively interested in the disease believe that to have made possible so sudden and destructive an outbreak there must have been some environmental change of major importance.[3] Environment in this case must mean, of course, a change in the sea water itself. Certainly this disease outbreak was no isolated phenomenon in North Atlantic water. It was accompanied, for example, by a marked decline in the catch of marine fisheries of New England, a decline from which it took five years to recover.

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[1]This paper was presented by R. B. Stevens at a symposium on the wasting disease held at the final session of the phytopathology section of the Seventh International Botanical Congress, Stockholm, Sweden, July 1950. It represents in summary form the results of some seventeen years of research by the senior author, ably assisted in the later phases by Miss Hazel Ellis, and from time to time by R. B. Stevens. After Neil Stevens' death in June of 1949, Miss Ellis continued the field work for one more summer, and did much to complete preparation of the manuscript. Ill health made it impossible for her to present the paper in Stockholm herself.

[2]Discussion of the paper brought out the fact that no cultural proof of the causal role of Labyrinthula has been offered; in part no doubt due to the difficulties encountered thus far in attempting to grow the suspected parasite in culture.

[3]Several participants in the discussion, particularly Scandinavian botanists, seemed in general agreement as to the likelihood of some such event. They pointed out that certain other marine plants, e. g. Codium, showed marked fluctuation in Baltic waters at about this time.

Fourteen years ago Stevens called attention to the fact that the outbreak of the wasting disease in the North Atlantic coincided with the occurrence of a "transgression" in the Atlantic Ocean. Since a transgression is in turn influenced by the position of the moon, the reaction to this suggestion was chiefly one of amusement. Apparently tide is the only phenomenon in connection with which the moon may be seriously mentioned.

Whatever the cause, we now know that the year 1931 was one of unusually high density in the Atlantic coastal waters of North America. Figures published by the U.S. Coast and Geodetic Survey show that at their tide stations there was a measurable and marked change in density. There are now available records of density and surface temperature of sea water from 1922 or 1923 to date at six stations within the normal range of eelgrass. The readings at Baltimore, Philadelphia, and New York are, of course, much influenced by great rivers near the mouths of which the stations are situated. The Boston station is somewhat better in this respect. Least influenced by fresh water from the land is Atlantic City, New Jersey, and next best is Portland, Maine. Both of these latter stations clearly show that the great outbreak of the wasting disease came during a period when the density of the sea water was well above the average and decidedly above that of five or six years earlier. This relationship shows distinctly in August, the month when the wasting disease usually strikes, and is great enough to affect the annual means. While the differences in density are admittedly small, the ocean is so large that any such general change in density is worthy of note. Moreover, students of marine biology are essentially unanimous on the point that the ocean environment is so stable that the physiology of marine organisms in general is easily disturbed by slight change. We have little experimental proof that this condition affected either Labyrinthula or its host, but in discussing so striking a phenomenon as the one under consideration such a condition should not be passed without mention. If such a relation exists, it seems probable that it is due to a change in salinity. Supporting these data, it should be mentioned that during the great outbreak of the wasting disease one of the most frequently observed facts was that Zostera persisted longest in areas of reduced salinity and made the first and most favorable return in such areas.

Addy states that in 1934 the disease was again severe in eastern Massachusetts on the rather limited stands which had reestablished themselves. And again this was a period of water density somewhat above average, at least near Boston.

The effect of temperature upon wasting of eelgrass has not been clearly shown. Field observers often note that symptoms of disease become marked as the waters warm. Young (1943) states: "The disease becomes rampant from mid-summer on, when coastal waters reach 16° C. or higher, depending on locality." Cottam and Addy (1947) observe that during July and August the organism is in greatest abundance and activity. As soon as cool weather begins, the activity of Labyrinthula drops rapidly until the organism is nearly dormant and much reduced in numbers during the winter. They state: "Temperature changes certainly exert a profound influence upon the activity of the disease organism."

Recovery

In studying the recovery of Zostera marina, it should not be forgotten that we are dealing with a plant considered monoecious, but which because the two sexes of flowers on a spathe rarely mature at the same time is essentially dioecious. Thus it has the maximum degree of heterozygosity and potential variability possible in a seed plant. This may (one can never know the facts) well have saved the species from extermination on the Atlantic coast. Certainly there was a period of at least eight to ten years when it was a serious question whether eelgrass would ever again be abundant there. Renn truly wrote in 1937, "Each spring since the extensive epidemic wasting of the common marine eelgrass. . . . . , five years ago, there have been anxiously optimistic reports of its reappearance at numerous points." This was the opening sentence of his report on conditions as observed during June 1936 in shallow bays from Greenwich, Rhode Island to Great Bay, New Jersey. Renn records numerous local observations and concludes with two generalizations: the Labyrinthula was present without exception in plants showing spotting or streaking; seedlings were scarce in foliated areas, indicating that the growth occurred chiefly from persistent stem stocks.

Fish and Wildlife Service reports---To Clarence Cottam and his associates in the Fish and Wildlife Service we are indebted for our best records of the condition of eelgrass during the critical decade following 1932. Summaries of field observations along the Atlantic coast of the United States were published by him for 1933, '34, '35, '37, '38, '40, '45 and '47.

A careful review of Cottam's reports shows clearly that in the earlier years many of the

plants seen were new growths from old rhizomes. In 1934, '35, '37, the condition of the plant continued extremely variable even where the environments appeared to be similar in regard to salinity, soil, and water depth. Symptoms of the wasting disease were often noted and on many occasions some areas were reported as decidedly worse than they were six to twelve months earlier. Cottam mentions the northern part of Chesapeake Bay, where salinity was markedly reduced, as showing the greatest promise of an early return. In general, also, progress was encouraging from Long Island Sound northward. Usually, in any given year, it seemed to the observers that considering the area as a whole conditions continued to improve. In his summary for the fall of 1940 Cottam said: "In sections of Chesapeake Bay conditions seem almost normal and in units of Pamlico Sound and localized portions of coastal New England the plant is perhaps ten to fifty percent as abundant as it was before 1931."

Certainly no one who understands the difficulty of field work, or the nature of estimates, or their importance in such a study, will be disturbed because the actual figures given by various workers are often at variance.

Even so late as 1945 Cottam reported: "Along most of the Atlantic coast of the United States and Canada the situation is now somewhat better than it has been since 1931. Local units may be called fully recovered; other areas still are almost completely without eelgrass." Commenting on his earlier observations that the condition of eelgrass improved first in areas of reduced salinity, he said: "Though improvement was first noticed in areas of reduced salinity, the condition is highly variable, in some areas of almost unmodified sea water, eelgrass has made an almost complete recovery, and in some areas of reduced salinity the situation is still most discouraging."

The most recent summary of the Fish and Wildlife Service report on eelgrass on the Atlantic and Pacific coasts was made in the summer of 1949. It shows a continued improvement in general and recovery of former abundance in some areas. Certain locations in Chesapeake Bay, Long Island Sound and Buzzards Bay have shown vigorous return. Coincident with this encouraging report Shuffler, reporting for Long Island, says, "The blue claw crab has also increased and there are more present than at any time in the past thirty years. Shrimp have also increased and local fishermen believe it due to the return of eelgrass." Regarding Pamlico Sound Cottam reported in November, 1949: "Without question, there is a marked improvement over conditions that existed there many years ago."

Other reports--For certain small areas detailed reports are available from observations made by several ecologists in connection with studies of the abundance of various invertebrates. Dexter writes as follows regarding conditions at Cape Ann, Massachusetts: "Between 1933 and 1940 the colonies increased yearly, growing chiefly from old rhizomes; most of the plants were diseased and neither survived for long nor led to a significant return of the species."

Stauffer, reporting on the invertebrates of the Northwest Cutter Lagoon near Woods Hole, states that the shallow water area had been changed from a dense matting of eelgrass to a vast extent of muck without any plant covering.

Dreyer and Castle in studying the occurrence of the bay scallop (Pecten irradians) at a number of localities on the coast of Long Island, made collections annually in August during the six years 1935-1940. They note the presence of eelgrass in "small bunches irregularly at various places."

By 1943, more than ten years after the great outbreak of the wasting disease, eelgrass was sufficiently abundant along the coast of Massachusetts to warrant a reconnaissance of its coastal waters. This work was undertaken by Addy and Aylward. The map published at that time gives a rough estimate of the relative abundance of the plant in various localities. The statement is further made: "The plant again may be exerting a favorable influence on water fowl." In spite of the gradual increase, Addy noted that eelgrass along the Massachusetts coast has shown widespread blackening and degeneration characteristic of the disease.

From a summary by Cottam and Addy, which was presented in February 1947 at the Twelfth North American Wildlife Conference, it is evident that during 1946 eelgrass continued to spread along the Atlantic coast, though the increase of course was by no means uniform. For example, on page 8 they write: "Undoubtedly the most extensive stands anywhere along the coast are located in Chesapeake Bay." They quote from Mr. F. M. Uhler, biologist of the Fish and Wildlife Service, who completed an extensive survey of the area January 7, 1947 as follows: "Since last April (1946) the recovery throughout the Bay has been widespread and in many of the estuaries and shoals this has been almost as spectacular as was the destruction of the plant in 1931." Addy found the plant in Massachusetts in excellent condition during late July and August. He says: "The health, vigor, and density of the beds were in striking contrast to the degenerate

appearance of those beds in 1944." The next year in a summary prepared by Addy and Robert S. Johnson the situation is summarized as follows: "The story of the steady increase of eelgrass the past year all along the coast is much the same as in 1946." With specific reference to Chesapeake Bay they say, "The principal areas consisting of many thousands of acres are located along the east shore of Maryland from about the Virginia line north to the vicinity of Baltimore."

Our own field observations, like those of Dexter's, supplement those of Addy and Aylward as they are confined to small parts of the Massachusetts coast, chiefly Upper Buzzards Bay. Almost all of the eelgrass in this area died during the summer of 1932. In September of that year the shallow waters of Buttermilk Bay, including Little Buttermilk, were so filled with floating remains of dead and dying eelgrass that no healthy plants could be found. A survey in this bay was, however, made by Stevens in the spring of 1933 and at intervals as opportunity offered during the next ten years. The results of some of these surveys have already appeared in the Plant Disease Reporter. All showed varying but small amounts of eelgrass. No doubt many of the clumps noted, such as those at the mouth of Red Brook, were growing from rhizomes which had survived the outbreak in 1931-32. As far as Stevens' observations go, at no time and in no place were seedlings abundant during this period. In 1945, when for the first time since 1932 floating masses of eelgrass were found in the waters of Buttermilk Bay, they later proved to have drifted in from the new stand on Hog Island Dike. The year 1946, a year of marked increase all along the coast, was the first year of really significant increase in growth of eelgrass in Buttermilk Bay.[4]

All available information regarding eelgrass on the Atlantic coast of North America from 1933 to 1949 agrees that its recovery has been slow and irregular. Beds have been reestablished for a time, only to be destroyed. There has been no observable relation between the rapidity of return and any known water condition. The behavior of eelgrass during this long period is apparently best explained on the assumption that there was going on a severe selection among seedlings, with the probable result that a number of relatively resistant strains have arisen and survived in different places and at different times. The pathogen apparently continued abundant and conditions were sufficiently favorable to assure the death of many plants. Judged by much less severe epidemics known among land plants, seedlings of adequate resistance might well arise wherever seeds were produced in fair abundance.

Some Factors that Affect Recovery

Reproduction -- Under many conditions Zostera marina is able to reproduce either vegetatively through the branching of rhizomes or by means of seeds. Observers seem not, however, to have any clear idea as to the relative importance to the plant of these two types of reproduction. No doubt this varies under different environmental conditions. Setchel, for example, suggests the possibility that eelgrass may have survived in the vegetative condition for a very long time on the west coast of Greenland. On the other hand, he does note that the flowering stem is an annual and that Zostera marina, while properly a perennial, behaves much like a biennial. All authorities agree that the plant is usually proterogynous and thus mostly cross pollinated. While both staminate and pistillate flowers occur in the same spathe, stigmas usually mature and drop off before the anthers.

In general, seedlings tended to be more abundant in somewhat northern water, that is north of Long Island Sound. If Setchell's observations are correct, reproductive activity goes on only during the period within which the temperature of the water is between 15° and 20° C., chiefly, if not solely, while the water is cooling to 15° in localities where the temperature seldom if ever exceeds 20° C. Surely though, in those localities where the mean water temperature does not reach 20° C. the total amount of seed production would be influenced by the length of time the water is rising from 15° to 20°. This is somewhat less than a month at Solomons Island, Baltimore, and Philadelphia, slightly more than a month at Atlantic City and Sandy Hook.

In the light of recent observations, it appears that either eelgrass does produce seeds below these limits of temperature or else our information on temperatures in Hudson Bay are false. Ellis found seedlings of eelgrass on the west side of Hudson Bay at about parallel 59° in July 1949. J. L. Tremblay reported that he saw good crops of eelgrass in three places on the east side of Hudson Bay as far north as parallel 60°. His observations were made in the summer of 1949. This information came as a surprise since it has been stated that eelgrass is limited in

[4] A short survey in August of 1950, following the Stockholm Congress, indicates that eelgrass has very nearly reestablished itself at the pre-epidemic level in Buttermilk Bay. Little Buttermilk, to the contrary, has little or none, and the present bottom is very silty and unstable. RBS

its growth to waters which reach a temperature of 8° C. or more during the summer.

Among the few observations of the recovery of eelgrass by means of seeds are those made in Upper Buzzards Bay by Stevens. In rebuilding the Cape Cod Canal in 1936-1937, sand was pumped up from the bottom of Buzzards Bay. The dike thus consists of new sand, that is, sand which has not been in shallow water for many years, probably in historic time. Yet on both the north and south dikes are healthy stands of eelgrass, stands which are among the best seen during the entire summer (1946). These stands could have come only from seed, probably from seed recently ripened on plants which survived the epidemic (in near-by coves); far less probably from seed that had lain on the bottom of the bay for years.

Resistance to Disease -- There has been to our knowledge no experimental work done to determine the existence of resistant varieties of eelgrass. Stevens did state in 1948 that, if since 1931 there had been underway an extensive program in breeding eelgrass for disease resistance, someone would now be announcing the production of a highly resistant variety.

Temperature Changes -- As has been pointed out earlier in this paper wasting is most evident in July and August. This is evidently one factor that has retarded recovery. A new stand may make considerable growth early in the season and be wiped out in the later months of summer. This had been noted in several places in Buzzards Bay.

Physical Character of Soil -- Our observations in Buzzards Bay indicate that those areas where eelgrass used to be and where the plant has failed to return have changed in physical character. These shallow bays and coves have become more muddy. Shellfishermen in this area agree that when eelgrass died out the bottoms became unstable. This area has been visited by two hurricanes since 1931 which are said to have buried the top sand layer with the mud which was beneath in those waters where there was no eelgrass. Observers find that seed beds require a layer of sand on top of mud; therefore, the only way these areas can be reestablished with eelgrass is by vegetative invasion. Although drifts of eelgrass with seeds are brought in by the tides, the unstable, muddy bottoms do not offer proper conditions for seed germination. Our tests have shown that there is no oxygen in the mud except on the very surface where conditions are so unstable that seeds become buried before they germinate or seedlings uprooted before they get established.

Cottam (1949) made similar observations. His report states: "Where once there was a dense growth, it is now found that the bottom, or beds have changed. Where once was a very favorite spot for growth of the grass, now is a very coarse gravel covered area. This change may be attributed to the continual dredging of channels and anchorage coves, thus presenting unfavorable conditions for the return of eelgrass."

Commercial activities -- Carter, reporting for Pamlico Sound, North Carolina, is of the opinion that eelgrass will not return to that area because of the severe activites of long netters, shrimpers, crabbers, and oyster dredgers. In Buzzards Bay we have noticed that the activities of clamdiggers have prevented recovery, while "closed" areas in the same shallow waters show good growth. Shuffler gives supporting evidence from Long Island, New York. The same report says, in regard to the Gloucester and Cape Ann areas of Massachusetts, "With the very large increase of fish processing plants in this immediate locality, with their drains and sewers emptying into the harbors, it is very hard to say that the return of eelgrass has been retarded entirely by disease."

Competing species -- It has been our observation in Buzzards Bay that when Zostera gets established in sufficient numbers in an area, it takes over. Ruppia maritima seems to be the only significant competitor in those waters that are being invaded by Zostera. This is true in waters from Pamlico Sound north.

Cottam reports small amounts of Ruppia maritima and Potamogeton pectinatus in drifts of eelgrass in Chesapeake Bay and considerable amounts of Halodule and Ruppia in Pamlico Sound.

Summary

At present we can only say with certainty that the destruction of eelgrass was wholesale in 1931-1932, that the plant has been recovering slowly, that some localities have made almost complete recovery while others have failed entirely. Considering the entire range of Zostera on the Atlantic coast of the United States of North America, there is more eelgrass at present than at any time since the great die-off.

Whether eelgrass is now able to withstand the pathogen under the normal fluctuations of the environment depends largely on whether or not resistant strains have developed; unless of course there were a widespread reduction in virulence of the pathogen.

Since local conditions often mask the larger trends, it is decidedly important that observers over the total range of Zostera continue to make and report their observations.

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UNIVERSITY OF ILLINOIS AND THE UNIVERSITY OF TENNESSEE

SOME VECTORS, HOSTS, AND PROPERTIES OF DAHLIA MOSAIC VIRUS

Philip Brierley[1] and Floyd F. Smith[2]

In earlier studies by one of us (1) dahlia-mosaic virus (Marmor dahliae Holmes) was found transmissible by the green peach aphid (Myzus persicae (Sulz.)) but not by leaf-rubbing inoculation. The only host plants established were species of Dahlia, and the green peach aphid was the only vector demonstrated. More recent publications dealing with dahlia mosaic have added no other hosts or vectors. In England (3) and in Belgium (2) the virus of tomato spotted wilt (Lethum australiense Holmes) and the cucumber-mosaic virus (Marmor cucumeris Holmes) are found prevalent in dahlias, and K. M. Smith (3) considers that both of these viruses outrank dahlia-mosaic virus in importance in England. In the United States, tomato spotted wilt -- which induces most if not all of the ring and oakleaf patterns described (1) as dahlia ringspot, yellow ringspot, and oakleaf -- has become more prevalent in dahlias; but cucumber mosaic has not been reported in these plants.

MATERIALS AND METHODS: Dahlia mosaic was obtained from a commercial stock of Jersey's Beauty, a mosaic-tolerant variety of long standing. All test plants were grown in an insect-free greenhouse. A separate greenhouse served for culturing of insects and for insect transfer trials. In certain tests aphids from non-viruliferous colonies maintained on appropriate healthy plants were transferred to the virus-infected source plants where they were allowed to feed for indefinite periods before they were transferred to healthy plants. In other tests the non-viruliferous aphids were transferred to small petri dishes and deprived of food for two to four hours, then allowed to feed for 15 minutes on leaf pieces from the source plant before they were transferred to healthy plants. The aphids were caged on small succulent tip leaves of one growing shoot on each inoculated plant by means of an open-end glass vial. All transfers were by brush. At the conclusion of such tests the plants were freed from insects by dipping in nicotine soap solution before they were returned to the insect-free greenhouse for observation. No dahlia mosaic contaminations appeared during the three years covered by this study.

The test plants were of Unwin's Hybrid strain grown from one clon in order to avoid variability in symptom expression. To develop this clon, seed of this strain were planted, and 20 of the resulting seedlings were propagated by cuttings to provide 20 clons. One plant of each clon was inoculated with dahlia mosaic by Myzus persicae. After two months all the clons were discarded except the one that expressed the symptoms most clearly. The test plants from this clon were generally obtained from cuttings. Supplemental light was supplied during winter months to promote active vegetative growth of the test plants.

NON-PERSISTENCE OF DAHLIA-MOSAIC VIRUS IN THE VECTOR MYZUS PERSICAE: The relation of dahlia-mosaic virus to the vector Myzus persicae was studied in the hope of improving the efficiency of experimental transmission. In an early series of experiments conducted on dahlia, the selected seedling clon served as source of virus and as test material. In some trials the aphids were starved for two to four hours in petri dishes before they were allowed to feed on source dahlias; in other tests the aphids were transferred from colonies long established on mosaic plants with no such starvation period. The usual interval of feeding on the source plant after starvation was 15 minutes. The interval of feeding on test plants was varied from one hour to 144 hours in different tests, and in some tests the aphids were moved at stated intervals to fresh test plants. The data from some of the more significant tests are summarized in Table 1.

These data show that Myzus persicae acquires the dahlia mosaic virus from dahlia as well after 2 to 4 hours of starvation as when taken direct from a long habitation on infected plants, or even better. Brief feeding periods on the source material after a period of starvation appear to be as effective in acquiring the virus as long periods. Brief feeding periods of 1 to 3 hours on the test plants seem as efficient in transmitting the virus as long periods of 1 to 6 days. When aphids were transferred to fresh test plants at intervals the virus was transmitted in the first hour of feeding and sometimes in the second or third hour, but never after 3 hours. Ten aphids seem to transmit as efficiently as 15 or 25 in these trials. A considerable variation in the number of plants affected is attributed to effects of temperature and other seasonal factors on the vector and on the test plants, or to the

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Table 1. Influence of a prefeeding period of starvation and of various periods of feeding on dahlia source plants and on test dahlia plants, on transmission of dahlia-mosaic virus by Myzus persicae.

Test number	Prefeeding starvation (hours)	Feeding periods: On source plant (minutes)	Feeding periods: On test plants (hours)	Aphids in test (number)	Plants infected [a]
6-994	2	15	144	25	4/5
6-996	None	2520	144	25	2/4
6-1027	2	15	First 2 hrs.	10	3/5
			Next 72 hrs.	10	1/5
6-1029	None	5880	First 25 hrs.	10	2/5
			Next 48 hrs.	2-4	0/5
6-1048	2	15	First 3 hrs.	10	2/5
			Next 19 hrs.	10	0/5
			Next 72 hrs.	8-10	0/5
			Next 96 hrs.	1-7	0/2
6-1049	4	10	96	15	9/10
6-1050	4	180	First 23 hrs.	10	4/5
			Next 68 hrs.	3-10	0/5
			Next 96 hrs.	8	0/1
6-1054	4	15	First hour	15	1/5
			Next 20 hrs.	15	1/5
6-1056	2	15	72	15	3/6
6-1057	3	15	First 1 1/2 hrs.	15	6/7
			Next 1 hr.	15	1/7
6-1062	2	15	48	25	4/5
6-1065	2	15	First hour	15	4/5
			Next 2 hrs.	15	3/5
			Next 23 hrs.	15	0/5

[a] Number of plants infected over number exposed.

succulence of the plants or their attractiveness to the aphids.

More recently, experiments on mode of transmission by Myzus persicae were conducted with Verbesina (Verbesina encelioides (Cav.) B. & H.) and Zinnia, (Zinnia elegans Jacq.). As discussed later these hosts were found to be better sources of the virus and better test plants than dahlia. In these experiments (Table 2) starved aphids transmitted the virus after an infection feeding period of 1 minute and a test period of 5 minutes. High transmission was obtained with 5 to 10 aphids with a 5-minute infection period and test periods of 5 to 15 minutes. Aphids transferred to fresh plants at intervals lost their ability to infect zinnia and Verbesina after the second hour. However, when aphids were starved for 3 hours after the infection feeding period then allowed to feed on Verbesina they were still efficient vectors (Table 2). The numbers of infected plants of zinnia and Verbesina were more uniformly high than in tests with dahlia.

It is clear from these tests that Myzus persicae carries the dahlia-mosaic virus in a non-persistent manner. Experimentally, a brief period of starvation of 2 to 4 hours, an infection feeding period of 10 to 15 minutes, and a test feeding period of 2 to 3 hours yield efficient transmissions in dahlia. In Verbesina and zinnia, a 1-minute infection period followed by a 5-minute test period resulted in infection. The transmission to dahlias in the third hour after the infection feeding period and only in the second hour on zinnia and dahlia may be correlated with less feed-

Table 2. Influence of time of feeding on Verbesina source plants, and on test plants of Verbesina or Zinnia, on transmission of dahlia-mosaic virus by Myzus persicae.

Test number	Prefeeding starvation (hours)	Feeding periods: On source plant (minutes)	Feeding periods: On test plants[a]	Aphids in test (No. per plant)	Plants infected[b]
6-1104	2	1	Overnight	5	1/5
		5	Overnight	5	3/5
		10	Overnight	5	3/5
		15	Overnight	5	3/5
6-1108	2	1	2 hours	10	5/5
		5	2 hours	10	5/5
		10	2 hours	10	5/5
		15	2 hours	10	5/5
		5	5 min.	10	5/5
6-1105	3	15	First hour	10	2/2
		15	2nd hour	10	1/2
		15	3rd hour	10	0/2
		15	4tn, overnight	10	0/2
6-1106	2	15	First hour	15	4/4
		15	2nd hour	15	1/4
		15	3rd hour	15	0/5
		15	4th hour	15	0/4
		15	5th, overnight	15	0/4
6-1107	3	15	First 15 min.	15	5/5
		15	2nd 45 min.	15	5/5
		15	2nd hour	15	2/5
		15	3rd hour	15	0/5
		15	4th hour	15	0/5
		15 min.--starved 3 hrs.--test feeding 3 hours		10	4/5

[a] Zinnia used in tests 6-1104, 6-1105, Verbesina in other tests.
[b] Number infected over number exposed.

ing of aphids on dahlia, an unfavorable host. The longer retention of virus by starved aphids supports this viewpoint. Practically, the short duration of retention of the virus by M. persicae suggests that dahlia seedlings isolated from disease sources by a few hundred feet should remain free from mosaic. This is in agreement with our experience with seedling dahlias in garden plantings.

ADDITIONAL VECTORS OF DAHLIA-MOSAIC VIRUS: When dahlia-mosaic virus proved to be carried in the non-persistent manner by Myzus persicae, it seemed likely that other vectors might be found, inasmuch as vector specificity is low among the non-persistent viruses. Accordingly five additional species of aphids were tested as vectors of this virus, using the techniques found effective with M. persicae. These trials were not made concurrently, but extended over a period of two years. Variations in the proportion of plants infected therefore should not be emphasized.

Aphis fabae Scop. transmitted dahlia-mosaic virus in 2 of 5 trials, infecting 4 of 51 plants. Aphis gossypii Glover transmitted in one test and failed in another, infecting 1 of 10 plants. Aphis maidis Fitch failed in one test on 5 plants. Macrosiphum solanifolii Ashm. proved an efficient vector in a single trial, infecting 6 of 10 plants. Myzus convolvuli Kltb. transmitted in one test but failed in another, infecting 1 of 18 plants.

Myzus convolvuli induces a direct feeding injury in dahlias that might be confused with dahlia mosaic in very early stages. Yellow angular spots appear at feeding sites after a few days. These spots persist, with more or less distortion of the adjacent areas of the leaf blade; but spots do not appear in new growth developed after removal of the aphids. True dahlia-mosaic symptoms in this seedling clon appear later, after 2 to 6 weeks, as slight downward curling of young leaves, followed by yellowish bands of varying width along the midrib and branch veins.

ADDITIONAL HOSTS OF DAHLIA-MOSAIC VIRUS: The dahlia seedling clon selected for these studies develops distinctive symptoms when infected, but for experimental purposes is undesirably slow to respond. It was thought that some related plant might prove better for test purposes. Accordingly, seedlings of a number of species of Compositae that were available in connection with other work were inoculated with dahlia-mosaic by means of the green peach aphid. Of the 14 species of plants tested Sanvitalia procumbens Lam.,[3] Verbesina encelioides, and Zinnia elegans (Lilliput types) developed mosaic. No symptoms appeared in Cichorium endivia L. (endive), Coreopsis lanceolata L., C. douglasii (Gray) Hall (received as Leptosyne stillmanii), Cosmos bipinnatus Cav., Cynara cardunculus L. (cardoon), Doronicum caucasicum Bieb., Helianthus annuus L. (var. Sun Gold), Lactuca sativa L. (Slobolt lettuce), Rudbeckia serotina Nutt. (received as R. hirta L. var. aurea), R. serotina f. pulcherrima (Farw.) Fern. & Schub. (Herbstwald var.), or Tithonia rotundifolia (Mill.) Blake. Dahlia mosaic was recovered in dahlia on sub-transfer of Myzus persicae from Coreopsis douglasii, Sanvitalia, Verbesina, and Zinnia, but was not recovered from the other plants named on similar sub-transfers. Thus C. douglasii was the only symptomless carrier detected in this limited sampling. The four additional plants found susceptible to dahlia mosaic are members of the tribe Heliantheae of the Compositae, as is Dahlia. Six of the ten plants that failed to take dahlia mosaic in these tests are also members of this same tribe.

Symptoms of dahlia mosaic in Zinnia appeared first as downward curling of young leaves, followed by yellowish narrow bands along some of the veins and marked dwarfing. Symptoms appeared first after 14 days and 7 of 10 plants were affected in one test, 2 of 10 in a second trial. In later stages after 2 months no mottling was evident, but the downward curling of the leaves and general dwarfing were conspicuous. In Verbesina symptoms appeared in 10 days as downward curling of young leaves. Vein clearing was evident after 10 days to 2 weeks (Fig. 1). In all, 6 of 9 plants developed symptoms. After one month the principal symptoms were broad bands of yellow along the midribs and branch veins. After two months this pattern was reversed, the previously yellowish band appearing as dark green and the rest of the leaf dull yellowish green (Fig. 1). There was considerable dwarfing of these plants. In Sanvitalia downward curling of young leaves, inconspicuous mottling, and general dwarfing were noted. No symptoms were detected in Coreopsis douglasii. In general symptom expression in these allied genera was similar to that in dahlia.

There appears to be no record of disease of the dahlia mosaic type in Zinnia in nature, although zinnias are widely grown. Verbesina and Sanvitalia are less common and may appear near Dahlia less frequently. It is not expected that dahlia-mosaic will prove troublesome in these annuals in gardens, inasmuch as the aphid vectors are infrequent in dahlias in gardens until late summer.

TRANSMISSION OF DAHLIA-MOSAIC VIRUS BY LEAF-RUBBING: Attempts to transfer dahlia-mosaic from dahlia to dahlia, by leaf-rubbing with carborundum dusted on the leaves to be rubbed, succeeded in three of four trials, 1/10, 1/20, 0/20, and 1/10 plants responding. When additional host plants of dahlia mosaic were detected by aphid inoculation, these were inoculated manually in the hope of finding a test plant more sensitive than dahlia.

These tests (Table 3) showed that Verbesina supplies a good source of virus and is sensitive to inoculation with dahlia-mosaic from Verbesina, although less sensitive to virus prepared in juices of the other susceptible plants. Verbesina is preferable to Zinnia for property studies in that it expresses more distinctive symptoms and also offers thin flat leaves that are readily inoculated and readily extracted. The results in Table 3 suggest, however, that Zinnia may be preferable as a test plant for indexing Dahlia or other hosts for this virus.

NON-TRANSMISSION OF DAHLIA-MOSAIC VIRUS TO CUCUMBER AND OF CUCUMBER-MOSAIC VIRUS TO DAHLIA: Inasmuch as the viruses of dahlia mosaic and of cucumber mosaic have aphid vectors in common, as both viruses are carried in the non-persistent manner, as

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[3]We are indebted to S. F. Blake for identifying the plants named in this paper.

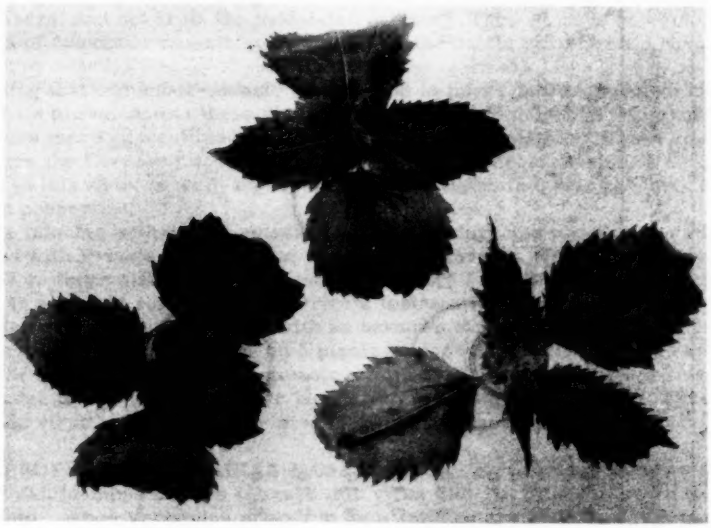

Figure 1. Symptoms of dahlia mosaic in Verbesina encelioides. Lower left, vein clearing after 2 weeks; lower right, broad dark green bands after 10 weeks. Upper plant is healthy.

Table 3. Comparison of the known hosts of dahlia mosaic as sources of virus and as test plants.

Source of virus	Plants inoculated		
	Dahlia	Zinnia	Verbesina
Dahlia	1/10[a]	3/9[a]	1/10[a]
Verbesina	7/10	8/9	9/10
Zinnia	0/10	8/10	1/10
Sanvitalia	----	8/20	2/10
Coreopsis douglasii	----	6/10	1/10

[a]Plants infected over plants inoculated.

both are juice-transmissible, and as cucumber mosaic is found commonly in dahlia in Europe (2, 3), it seemed desirable to reexamine the question of their possible identity. Tests of transmission of dahlia-mosaic virus to cucumber were made with Myzus persicae. In one test 1 of 5 dahlia plants was infected, and none of 5 cucumbers. In a second trial 4 of 5 dahlias developed mosaic, but none of 5 cucumbers. Thus no infection was produced in cucumber with dahlia-mosaic virus.

Leaf-rubbing inoculations of cucumber-mosaic virus into seedling dahlias of the Coltness Hybrid type were made 12 years ago in connection with other studies. Four strains of the virus originally from vegetables and maintained by S. P. Doolittle, and one strain from Easter lily were included in this trial. The vegetable strains included Doolittle's type, Price's type, and Wellman's celery strain. Each of the 5 cucumber-mosaic strains was inoculated into 10 dahlia seedlings. No symptoms resulted and no virus was recovered from any of the five sets of dahlias on subinoculation to tobacco. Recently Doolittle's type strain from tobacco was again tested in 20 dahlia seedlings of the Unwin's Hybrid clon mentioned earlier in this paper. No symptoms appeared and the virus could not be recovered in tobacco from the inoculated dahlias. In tests with the vector Myzus persicae Doolittle's type strain was transmitted to 5 of 5 cucumbers but to

Table 4. Physical properties of dahlia-mosaic virus determined in seedlings of Verbesina encelioides. Results of two tests are given.

Thermal inactivation			Dilution tolerance			Aging tolerance			Drying tolerance		
Unheated	10/10[a]	10/10[a]	Undiluted	9/10[a]	10/10[a]	Fresh	9/10[a]	6/7[a]	Fresh	9/10[a]	6/7[a]
50° C.	4/10	--	1:10	6/10	9/10	1 day	8/10	--	7 days	6/10	6/7
55° C.	7/10	--	1:30	1/10	9/10	2 days	10/10	--	10 days	0/10	7/7
60° C.	2/10	8/10	1:100	1/10	3/10	3 days	4/10	6/7	14 days	0/10	0/7
65° C.	4/10	10/10	1:300	0/10	2/10	4 days	3/10	--			
70° C.	7/10	10/10	1:1000	0/10	2/10	7 days	8/10	2/7			
75° C.	--	9/10	1:3000	0/10	1/10	10 days	7/10	7/7			
80° C.	--	10/10	1:10,000	0/10	--	14 days	9/10	6/7			
85° C.	--	1/10				17 days	--	2/7			
90° C.	--	0/10				21 days	--	5/7			
						28 days	--	4/7			
						35 days	--	0/7			

[a]Plants infected over plants inoculated.

none of 5 dahlias. The virus was readily subinoculated to tobacco by leaf-rubbing from the inoculated cucumbers, but not from the inoculated dahlias. Thus we have no evidence that American strains of cucumber mosaic virus can infect the dahlia varieties we have used as test plants.

It is surprising that cucumber-mosaic virus failed to infect dahlias here, in view of recent reports (2, 3), of its prevalence in these plants in Europe. It is possible, of course, that other types of dahlias are more susceptible to this virus than the Coltness and Unwin types we have used. Or, perhaps the European workers are dealing with different strains of cucumber-mosaic virus, inasmuch as this virus is well-known to be differentiated into many strains that differ in host range and in properties.

Zinnia plants infected with dahlia-mosaic virus for 24 days and showing systemic symptoms were reinoculated with Price's tester strain of cucumber-mosaic. Ten brown spreading primaries developed on these plants and the same number on 10 healthy zinnia plants inoculated at the same time. In a second series of tests Price's tester strain was inoculated into zinnias previously infected with dahlia-mosaic or with an unknown virus isolated from Impatiens balsamina L. No primary spots developed on 5 plants previously infected with the unknown virus, which proved to be a strain of cucumber-mosaic virus, but 9 plants previously infected with dahlia-mosaic developed 20 to 50 spots per plant from the tester inoculation. These data show that dahlia-mosaic virus is not closely related to cucumber-mosaic virus.

PHYSICAL PROPERTIES OF DAHLIA-MOSAIC VIRUS: No previous data are availabe on the properties of dahlia-mosaic virus because this virus was not hitherto known to be mechanically transmissible. When Verbesina proved to be a satisfactory test plant it became feasible to determine the chief physical properties of the virus. Juices of Verbesina are readily extracted after freezing for 72 hours at -5° C., but such an extract served to infect only 6 of 20 seedlings, whereas juices extracted in a mortar with a minimum of water (about 2 parts water to 1 part leaves by weight) infected 9 of 10 Verbesina seedlings in a parallel test. Juice was prepared in the latter way for thermal inactivation, dilution tolerance, and aging tolerance determinations. In aging tests 2-cc. samples of crude extracted sap were stored in open test tubes at 18° C. For the drying tests leaves were allowed to dry on a greenhouse bench, and ground with a little water for test after the intervals stated. The results are shown in Table 4.

The thermal inactivation end point is found to lie between 85° and 90° C.; the dilution end point is above 1 in 3000. The virus remains infectious in drying leaves for 7 to 10 days, or until the leaves are nearly air-dry. Extracted sap stored in open tubes at 18° C. remains infectious after 28 days, not after 35 days.

SUMMARY

1. Dahlia-mosaic virus is carried in the non-persistent manner by Myzus persicae. The virus is acquired in a 1-minute feeding period, and persists in this vector for 3 hours or less.

2. Aphis fabae, Aphis gossypii, Macrosiphum solanifolii, and Myzus convolvuli also transmitted dahlia-mosaic virus, but Aphis maidis failed to do so in a single trial.

3. Sanvitalia procumbens, Verbesina encelioides, and Zinnia elegans can be infected with dahlia-mosaic virus, developing symptoms similar to those expressed by dahlia. Coreopsis douglasii developed no symptoms when inoculated, but dahlia-mosaic virus was recovered.

4. Dahlia-mosaic virus is transmissible with difficulty from dahlia to dahlia by leaf-rubbing, but with ease from zinnia to zinnia and from Verbesina to Verbesina by this method.

5. Dahlia-mosaic virus was not transmitted to cucumber by Myzus persicae, nor was cucumber-mosaic virus transmitted to dahlia by this vector or by leaf-rubbing in our trials. Dahlia-mosaic failed to protect zinnia against infection by Price's tester strain of cucumber-mosaic virus.

6. Physical properties of dahlia-mosaic virus, determined in Verbesina, are thermal inactivation between 85° and 90° C.; dilution end point above 1 in 3000; infectious in drying leaves after 10 days, not after 14 days; and infectious in crude sap extract stored at 18° C. after 28 days, not after 35 days.

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CANTALOUPE MOSAIC AS AFFECTED BY NITROGEN FERTILIZATION

F. W. Zink and G. N. Davis

Investigations on the effect of cultural practices on cantaloupe mosaic have been directed toward the nutritional phase during the past two seasons. The results of the 1949 work indicated no response in yield to the heavy split application of nitrogen over the growing season. However, the infestation of mosaic in the 1948-1949 season was very late, with the results that the cantaloupe plants were well set with fruit, and satisfactory yields were obtained despite the presence of the virus. The purpose of the 1950 study was to determine:

1. Whether the application of nitrogen fertilizer above the normal amount through the growing season would alleviate the severity of the disease.
2. Whether such application of nitrogen would delay the maturity of the fruit.
3. Whether such application of nitrogen would affect the sugar content of the fruit.

PROCEDURE: The variety used was Powdery Mildew Resistant Cantaloupe No. 6. The seed was planted without hot caps on February 19, 1950, at the Imperial Valley Field Station, maintained by the University of California at El Centro, California. A randomized block design was used for this experiment consisting of 6 treatments with 4 replications.

Each treatment consisted of applications to a single bed 160 feet long, an area of approximately 1/45.3 of an acre. The 6 treatments are presented in the following table:

Treatment No.	Fertilizer material	No. of Applications	Date of application				Total pounds Nitrogen/Acre*
			1	2	3	4	
1	None						
2	Amm. Nitrate	1	2/19				60
3	Amm. Nitrate	2	2/19	3/20			60
4	Amm. Nitrate	4	2/19	3/20	4/10	5/10	120
5	Amm. Nitrate	4	2/19	3/20	4/10	5/10	180
6	Nitric Acid	4	2/19	3/20	4/10	5/10	60

*Total pounds divided equally between applications

All plots received a pre-planting application of treble superphosphate at the rate of 92 pounds of P_2O_5 per acre. This material was drilled in a band 6 inches under the seed. The ammonium nitrate was applied with a Fairbanks fertilizer drill at a depth of 3 inches along the side of the furrow approximately 6 inches from the plants. The nitric acid was supplied in the irrigation water with run-off at the end of the furrow held to a minimum. Records were taken of vine condition and severity of virus infection. Yield data from each harvest consisted of the number of marketable fruit, weight of marketable fruit, number of cull fruit, and classification of non-marketable fruit as to why they were culled. Five marketable cantaloupes were selected at random from each treatment on four consecutive harvests. Refractometer readings were taken of these melons to determine the percentage of soluble solids.

OBSERVATIONS: Symptoms of the virus were first noticed on March 17, and by the end of the month the field appeared to be 100 percent infected. The virus symptoms were quite severe in all treatments. Treatments 1 and 6 had the most pronounced symptoms and never recovered from the shock of the infection. By mid-June the plants in these two treatments had lost more than 50 percent of their foliage. There were no visual differences between treatments 2, 3, 4, and 5. All recovered slowly from the initial shock of the virus. However, at the end of the season these vines were stunted, and new growth showed leaf distortion and moderate chlorosis.

RESULTS: Analysis of the harvest records (Table 1) indicates that a singificant difference exists between treatments for marketable fruit, mean weight of marketable fruit, and the percentage of the total fruit harvested which was marketable. Treatments 3, 4, and 5 produced significantly more marketable fruit than treatments 1 and 6.

The application of nitrogen increased the mean weight of marketable fruit. Treatments 3, 4, and 5 are significantly greater than treatment 1, and treatment 5 was also significantly greater than treatment 6. The percentage of marketable fruit within each treatment was obtained by counting the number of marketable fruit present as against the total fruit ripening during the harvest period.

Table 1. Effect of nitrogen on marketable yield and quality.

Treatment [+]	Marketable yield per acre crates	Mean weight marketable fruit lbs.	Mean soluble solids %	Total fruit marketable* %
1 Check	99.2	2.17	11.71	63.9
2 60 lbs. N (1 application)	110.2	2.30	11.69	71.6
3 60 lbs. N (2 applications)	125.2	2.35	11.63	76.2
4 120 lbs. N (4 applications)	125.0	2.34	11.77	76.8
5 180 lbs. N (4 applications)	134.0	2.46	11.64	80.7
6 60 lbs. N (4 applications)	88.5	2.21	11.70	59.6
Least significant difference for odds of 19:1	25.6	0.15	N.S.	

[+]Ammonium nitrate used in treatments 2 to 5 inclusive. Nitric acid in treatment 6.
*Percentages were transformed to degrees percentage (p) = sign $^{2}\Theta$ for statistical analysis. Treatment 6 yielded a significantly lower percentage of marketable fruit than treatments 2 to 5 inclusive, while treatment 1 gave significantly less than treatments 3 to 5 inclusive.

Treatment percentages were then analyzed; and the same significant differences existed between the percentages of fruit marketable as in the total marketable fruit -- with the exception that treatment 2 gave a significantly greater percentage of marketable fruit than did treatment 6.

Analysis of variance for the percentage of total soluble solids content of fruit indicated that there were no significant differences between treatments.

An attempt was made to classify the culls. Four classes were established: 1. fruit that would be culled because of small size; 2. fruit that had size but would be culled for lack of netting (slicks); 3. fruit that had size but was sunburned; 4. fruit that had size but was injured (water rots, scuffs, and insect damage). Since all but a few cantaloupes showing sunburn injury were classified as slicks, this class is not shown in Table 2.

Table 2. Effect of nitrogen on total yield of fruit and on defects.

Treatment [+]	No. total fruit/acre	No. small fruit/acre	No. slick fruit/acre	No. fruit with injury/acre*
1 Check	7009	1259	1338.7	63
2 60 lbs. N (1 application)	6919	720	1102.5	78
3 60 lbs. N (2 applications)	7391	709	1080.0	49
4 120 lbs. N (4 applications)	7312	607	956.2	60
5 180 lbs. N (4 applications)	7683	551	1091.2	70
6 60 lbs. N (4 applications)	6682	1260	1462.5	79
Least significant difference for odds of 19:1	N.S.	471	317.2	

[+]Ammonium nitrate used in treatments 2 to 5 inclusive. Nitric acid in treatment 6.
*All fruit injured by wind, insects, and water rots.

The addition of nitrogen at the treatment levels used in this investigation had no effect on total set of fruit maturing during the harvest period. Nitrogen applications except treatment 6 significantly decreased the number of small fruits, compared to the check. With the heavier application of nitrogen there was a trend toward a reduction in the number of slick fruits. A significant difference in this respect existed between the 120 pounds nitrogen treatment and the no nitrogen treatment, or when part of the nitrogen was given in the form of nitric acid.

No significant effect resulting from the various nitrogen treatments was observed on the

maturity rate of cantaloupes. However, the no nitrogen check and treatment 6 reached peak production before any of the nitrogen treatments (Figure 1).

Some explanation should be made as to the poor showing of nitric acid. The results indicate that a considerable amount of this material was lost in run-off water or leached below the root systems of the plants.

Figure 2 demonstrates the percentage of cull fruits for each harvest over the growing season. The greatest proportion of cull fruits appears in the crown set. The principal cause of the large number of non-marketable fruits in the early set is small size. It will be noticed that the application of nitrogen reduced the percentage of culled fruit in the early harvest.

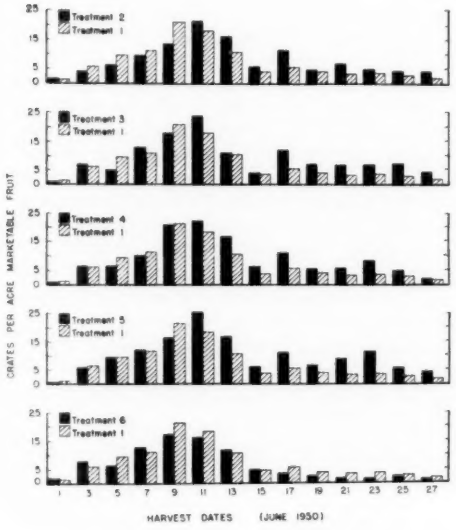

Figure 1. Crates of marketable fruit per acre (ordinate) for 5 nitrogen treatments and a check as a function of harvest dates (abscissa).

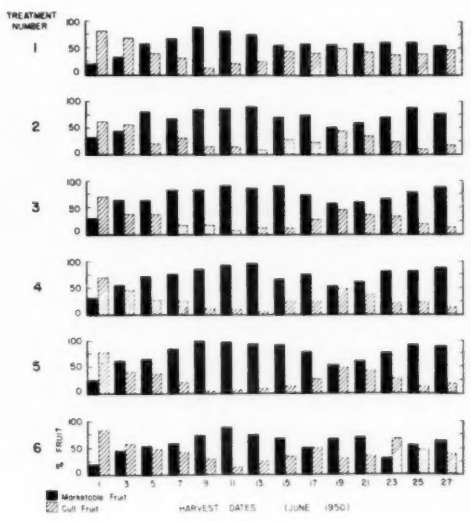

Figure 2. Percent marketable fruit and percent cull fruit (ordinate) for 5 nitrogen treatments and a check as a function of harvest dates (abscissa).

SUMMARY

The response of the cantaloupe plants to nitrogen fertilizer appears to be an increase in fruit size and a trend toward reduction in the number of slick fruits. No increase in the number of fruits set, total soluble solids of the fruits, nor retardation in the rate of maturity was apparent with the use of more than 60 pounds of nitrogen per acre.

These results substantiate the findings of the previous season that the severity of the mosaic infection as reflected in yield of marketable fruit is not alleviated by the heavy split application of nitrogen during the growing season. The supplying of 60 pounds of nitrogen, with half of it used as a pre-planting application and the remainder as a side dressing when the runners start, appears to be the most satisfactory nitrogen fertilizer program.

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LATENT RINGSPOT VIRUS OF CHERRIES REDUCES GROWTH OF NURSERY TREES[1]

J. A. Milbrath

Many of the sweet and sour cherry varieties which are being propagated in the United States carry one or more strains of the ringspot virus (1) in a latent condition. These can be detected by inoculation into Shirofugen and Kwanzan Flowering cherry which have been developed as index plants for this virus (2, 3). In developing a program for Oregon nurseries propagating cherry trees, it was noted that indexed, virus-free, sweet and sour cherry trees produced a better stand of nursery trees which were more uniform and larger than trees propagated from the regular run of nursery stock. In order to get actual measurements of this difference in growth rates an experiment was organized in cooperation with a local nursery, whereby direct comparisons could be made.

The experiment was laid out in a regular nursery block planting where a uniform age and size of mazzard seedlings had been used as liners for rootstocks. The major portion of the block was budded by the nurseryman in the normal manner of cherry propagation, but certain rows were reserved adjacent to each variety for buds from indexed trees. Royal Anne (Napoleon), Bing and Lambert were selected for comparison with the same 3 varieties used in the rest of the nursery block. Royal Anne, A10 and Bing, B260 are apparently free from the ringspot virus. Royal Anne, A114 was negative on Shirofugen and reacted mildly on Kwanzan indicating a mild strain of the ringspot virus. No virus-free Lambert was available. The Lambert, L89 used in studies reacted on both test plants but very mildly on Kwanzan, again indicating a mild strain of the ringspot virus. The Royal Anne, Bing and Lambert used by the nurseryman were infected with a severe strain of ringspot which reacted on both test plants.

The trees were budded in August and at the end of one growing season two hundred trees from each bud source were measured from the bud union to the tip of the growing terminal. Table 1 gives a summary of these measurements showing differences in average height and also the percentage of trees which were grouped in different height groups.

Table 1. Comparative rate of growth on tested virus-free nurserystock and average untested nurserystock.

Variety and tree code	Virus reaction		% of trees in each height group			
	Kwanzan	Shirofugen	5'-	4'-5'	4'-	Average height
Royal Anne A 10	-	-	48.7	34.2	17.1	4.6
" " A 114	Mild	-	31.6	46.9	21.5	4.4
" " A 7	Severe	+	4.8	53.3	41.9	3.8
Bing B 260	-	-	48.4	34.8	16.8	4.6
" B 386	Severe	+	0.2	33.1	66.7	3.3
Lambert L 89	Mild	+	45.1	27.7	27.2	4.3
" L 6	Severe	+	1.3	28.5	70.2	3.3

The differences between the growth of the virus free stock and the regular nurserystock were quite apparent without comparative measurements. All rows of virus free stock stood out as a unit and might easily be mistaken for a different variety. The data in Table 1 show that the virus free stock averaged from 8 to 14 inches taller than the same varieties carrying severe strains of ringspot, but the differences appeared even greater when the rows were compared in the nurseries. The mild strains of ringspot did not reduce the growth as much as the severe strains.

Of special interest are the two Bing sources. Both originated from the same source tree, but B386 became infected with ringspot virus when it was topworked on an infected mazzard

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seedling in a scion orchard. In this comparison we have the same clonal type. The only variant was the ringspot virus.

The same difference in the rate of growth has been noted for indexed, virus-free Montmorency sour cherry trees and those infected with the ringspot virus. No measurements have been made of these differences, but when the two stocks are grown in adjacent rows the contrast is quite striking (Figure 1). The trees are not as vigorous and the branches are 10 to 12 inches shorter. The stand of buds in a row of Montmorency nurserystock has been noted to be reduced as much as 50 percent by using scion wood containing the ringspot virus.

Figure 1. The effect of ringspot virus budwood on subsequent growth of Montmorency sour cherry nurserystock. The 3 trees on the left were propagated from infected buds and the 3 trees on the right were from virus-free buds.

An experimental orchard has been established at The Dalles, Oregon comparing virus free Royal Annes, Bings and Montmorency sour cherry with the same varieties carrying a severe strain of ringspot virus. The planting is replicated on Mazzard and Mahaleb rootstocks. Rates of growth and subsequent yields of fruits will be compared.

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OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

TWO VIRUS DISEASES OF BLUE LUPINE[1]

J. L. Weimer[2]

Two virus diseases of blue lupine (Lupinus angustifolius L.), seldom observed previously, were present in epiphytotic form in lupine fields at two locations in Georgia in the spring of 1949. In 1950 both diseases were present in numerous fields, but usually only an occasional plant was infected. Although a detailed study of these diseases has not been possible, the observations and few data at hand are presented herein.

One of these diseases was present in severe form in several experimental plots at the Georgia Coastal Plain Experiment Station, Tifton, Georgia, and the other on a farm at Oglethorpe, Georgia, in the spring of 1949. Up to 100 percent of the plants in some areas were diseased and many were killed. These diseases were present in trace amounts only, if at all, at the same locations in 1950.

SYMPTOMS: Both diseases caused dwarfing, malformation, and ultimately death of the affected plants. Plants exhibiting all stages of the diseases were present at the same time. Other symptoms of the two diseases were distinct. Plants affected by the virus from Tifton had greatly dwarfed and curled leaflets, especially towards the upper part of the plants. The tips of the leaflets characteristically curled down and back towards the lower surface of the leaflet (fig. 1). The leaf then had the appearance of a partly closed hand turned downward with the fingers, represented by the leaflets, half-closed or pointing towards the palm. The diseased leaflets were about one-half to two-thirds the width of healthy plants. The foliage of diseased plants was typically yellowish-green in color. Affected plants developed a large number of leaflets from axillary buds and branches.

The leaflets of plants affected with the disease from Oglethorpe were somewhat dwarfed, but were distinctly wider and thicker than those of plants affected with the virus from Tifton. Leaflets of the affected plants at Oglethorpe were rigid and pointed upward rather than down, so that the leaf resembled a partly closed hand with the fingers pointing upwards. (fig. 2). The lower surface of the leaflets often was somewhat purplish (Vandyke red of Ridgway) as contrasted with the yellowish-green of the leaflets affected with the virus from Tifton. In plants affected with either of these diseases the leaflets became smaller as the plants died.

The xylem, pith, and cortex of the stems and roots of plants in an advanced stage of either disease became brownish in color and death and decay finally resulted. Decay probably was at least hastened as a result of invasion by secondary fungi such as Alternaria sp. and Fusarium sp. The exterior of diseased stems and roots became dark brown. Discoloration started in the main root and then gradually spread up the stem and throughout the root system. Diseased plants produced few seeds, and those usually were small and shriveled.

ETIOLOGY: In 1949 the disease at Tifton was most prevalent where lupines were planted 50 feet or less from plots of Dixie Wonder peas (Pisum sativum), which showed mottling but little or no malformation. This suggested lupine and pea were at least common hosts of the virus and that this virus might be the pea mosaic virus. Additional evidence this might be the case was obtained in 1950 when it was noted that, when the Dixie Wonder pea was omitted from the rotation, no disease appeared in the lupine.

Diseased plants were obtained at Tifton and transplanted to pots in the greenhouse at Experiment, Georgia. Juice inoculations were made in healthy lupine seedlings using carborundum. The plants were inoculated on February 11, 1949. On March 19, 1949, infection was clearly evident. The first symptoms noted were dwarfing, an increase in the number of leaves along the stem, and a droopy appearance of the tips of the plants. There was no crooking in the terminal end of the stem, or streaking along the side of the stem as previously described for the disease of lupine caused by the pea mosaic virus. At this time the diseased plants were about two-thirds as tall as the control plants. On March 30 infected plants were one-half to two-thirds as tall as the controls, were yellowish-green in color, and had numerous small curled axillary leaves about one-third normal size. Some leaflets were curled so tightly they tended to form ball-like

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[1]Cooperative investigations of the Division of Forage Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture, and the Georgia Experiment Station. Paper No. 203 Journal Series, Georgia Experiment Station.

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Figure 1. Blue lupine plant at the left was an uninoculated control. The one at the right was infected with the virus from Tifton, Georgia as a result of juice inoculation. Inoculated February 25 and photographed March 28, 1949. The diseased plant was greatly stunted, the foliage greenish-yellow in color, and the leaflets were small and curled under.

Figure 2. A portion of a stem from Oglethorpe, Georgia, affected with what is considered to be a virus disease, although transmission attempts gave negative results. The leaflets are rigid and point upwards instead of curling downwards as do those in fig. 1

structures at the tops of the plants. There was slight mottling at this time, but it was not a conspicuous symptom.

On March 31, 1950 a number of blue lupine, Dixie Wonder pea, pepper (Capsicum annuum), white lupine (Lupinus albus), soybean (Glycine max), cucumber (Cucumis sativus), and tomato (Lycopersicum esculentum) plants were inoculated by the same method. Blue lupine, pea, pepper, and cucumber also were inoculated with the virus using aphids as vectors. The aphids were obtained from cantaloupes in the greenhouse, but the species was not determined. In some experiments infected pepper plants were used as the source of inoculum. The data obtained are summarized in Table 1. These data show the virus was transmitted by the juice method from blue lupine to blue lupine in six of the 30 plants inoculated. It was not transmitted from pepper to blue lupine. Infection was obtained in the two lupine plants in which aphids were used as vectors. What appeared to be infection was obtained in Dixie Wonder pea both by the juice method and with aphids, but similar symptoms appeared in some of the controls; hence these results must remain in doubt. The disease was transmitted from blue lupine to pepper by the juice method and with aphids. Transmission from blue lupine to cucumber took place readily by both methods of inoculation. None of the other plant species tested were infected. Since infection usually was obtained in a relatively small proportion of the lupine plants inoculated, the lack of infection in some of the other plants may be due to the small numbers used.

This virus was readily transmitted from blue lupine to blue lupine, pepper, and cucumber, and possibly to Dixie Wonder pea. It was not transmitted to white lupine, soybean, or tomato. In no case was the disease transmitted when pepper was used as a source of inoculum. In pepper the disease produced a definite mosaic with a faint line pattern and some malformation of the leaves. The characteristic symptoms in cucumber were vein clearing and mottling.

At this stage in the investigation the work had to be discontinued. The diseased plants soon died, and the source of the inoculum was lost. No final conclusions, therefore, can be drawn regarding the relation of this virus to other viruses.

The Oglethorpe disease was assumed to be caused by a virus since the symptoms were typical of those caused by viruses in other plants. They consisted of malformation of the new growth,

Table 1. The amount of infection in several hosts inoculated with the virus from Tifton by the juice method and with aphids.

Plants tested	Source of inoculum	METHOD OF INOCULATION: Juice: Number of plants: Inoculated	Juice: Number of plants: Control	Juice: Plants infected: Inoculated	Juice: Plants infected: Control	Aphids: Number of plants: Inoculated	Aphids: Number of plants: Infected
Blue lupine	B.L.[a]	30	12	6	0	2	2
	P[b]	2	0	0	0	0	0
Dixie Wonder Pea	B.L.	16	2	+[c]	+	5	2
	P.	5	0	0	0	0	0
Pepper	B.L.	11	19	3	0	1	1
	P.	2	0	0	0	0	0
Cucumber	B.L.	9	0	4	0	1	1
	P.	6	0	0	0	0	0
White lupine	B.L.	1	0	0	0	0	0
Soybean	B.L.	8	0	0	0	0	0
Tomato	B.L.	2	3	0	0	0	0

[a]Blue lupine
[b]Pepper
[c]Infection appeared in both the inoculated and the control plants in both experiments.

gradual dwarfing, and finally death of the affected plants. Microorganisms were absent in the early stages of the disease, but later invaded the tissue. Although the lupines were growing in a pecan grove there was no evidence the symptoms might have been caused by any spray material used on the pecans. All efforts to transmit the Oglethorpe disease by juice inoculations under greenhouse conditions were unsuccessful. When seeds from known diseased plants were planted in the greenhouse only healthy plants emerged. Most of the seed collected[3] were small, some were shriveled, and many failed to germinate; hence a small number of seedlings was obtained.

SUMMARY

Two diseases of blue lupine sometimes seen in commercial lupine fields were observed in epiphytotic form in the spring of 1949 at Tifton and Oglethorpe, Georgia. The disease appearing at Tifton was transmitted a number of times both by the juice methods and with aphids; hence its virus nature was demonstrated. Field evidence indicated the disease was caused by the pea mosaic or some other pea virus, since it appeared in severe form only in plots near Dixie Wonder field peas in 1949 and failed to reappear in 1950 when this pea was omitted from the rotation. The symptoms produced, however, did not check very closely with those described in the literature for the pea mosaic virus on lupine or any other virus disease of blue lupine described in the literature. Studies were not carried far enough to establish the relationship of this to other viruses. All attempts to transmit the disease found at Oglethorpe were negative. Neither of these diseases are thought to be seed-borne.

DIVISION OF FORAGE CROPS AND DISEASES, AND THE GEORGIA EXPERIMENT STATION

[3]Thanks are due to Dr. Glenn W. Burton for harvesting the seed at Tifton.

A REPORT OF GRAYWALL OR INTERNAL BROWNING OF TOMATO IN SOUTH FLORIDA

Warren N. Stoner[1] and William D. Hogan[2]

Losses suffered by some tomato growers during recent crop seasons from a condition known as graywall have caused several to consider discontinuing tomato culture. The condition was observed in fields from Vero Beach south to Pompano and west to Loxahatchee.

Symptoms of the Florida graywall condition are quite similar to a disease described by Haenseler (2) and Holmes (3) under the name of internal browning. A type of internal browning disease of tomato in New Jersey, according to Holmes (4) is caused by strains of tobacco mosaic virus recovered from Plantago species. Young (6) has also described a condition of tomato occurring in Texas under the name of internal browning or core rot, which in some cases closely resembles Florida graywall. Internal browning and core rot symptoms according to Young (6) probably have the same cause as blossom-end rot.

The symptoms of graywall observed by the writers agree for the most part with those described by Conover (1) as vascular browning, and it is believed the condition observed was the same. Some differences were noted in the symptom expression however. Uneven ripening was common and apparently conditioned in amount by the severity of the internal browning. Disintegration and browning was observed to extend into the placental and core tissues in many fruits. Generally the browning of the vascular tissues stopped before reaching the abscission point in the fruit trusses, but it has been seen in several instances to extend into the main stem running up and/or down for short distances (2 to 4 inches). These symptoms agree rather closely with those described by Young (6) and Haenseler (2). In the more severe cases a general disintegration has been observed in Florida.

The local name "graywall" is derived from the appearance of the condition in green and pink fruits. The internal browning shows as a pale to dark gray poorly defined area in the outer wall of the whole fruit. As a rule, these areas are seen in the outer wall of the locule but they can and do extend into and across the septa. As the fruit ripens, the color of the gray areas becomes brownish and portions of the wall surrounding the areas often fail to color properly. When the fruits are fully red ripe, the brownish areas are slightly depressed and roughened.

Research at the Everglades Experiment Station on this problem has been confined to attempts to determine its cause. Repeated microscopic examinations of field plants bearing graywall fruit have failed to disclose any visible organism. Since field observations to date have failed to show any relation to nutrition, the cause has been thought to be a virus. Holmes's (4) findings on internal browning of tomato in New Jersey, however, have not been applicable in Florida. No association of graywall in Florida could be made with Plantago species, no definite pattern of spread was observed, and no consistently recognizable foliage symptoms have been seen in plants bearing graywall fruit.

A field located at Loxahatchee, Florida, was found to be heavily affected with the graywall condition. Six graywall plants in this field were selected at random for source material. The plants in the row adjacent to the selected plants were pulled, and for the rest of the growing season the source plants were under the same cultural practice as the rest of the field, except the pickers were not allowed to harvest the fruit. All inoculation material used in the investigation was taken from these six plants, and each time material was collected one or more of the plants were bearing graywall fruit.

Mechanical inoculations by the carborundum method, described by Rawlins and Tompkins (5), were made during the spring of 1950 on the following dates: January 30, February 28 and March 9. Three plants each of tomato, Turkish tobacco, Nicotiana glutinosa, squash and cucumber were inoculated with sap from each of the six source plants each time. A total of 270 plants were inoculated over the three-month period. No local or systemic symptoms were seen in any of the rapidly growing inoculated plants. In each set all plants except for the tomatoes were discarded at the end of 30 days. The tomatoes were held for observation to see if fruit symptoms would develop. Fifty-four plants, nine inoculated from each source plant, produced 239 fruits. Each fruit was harvested when fully red ripe. All were cut in cross sections for internal examination and none of them showed internal browning.

The first and third time field material was collected for mechanical inoculation a sufficient number of scions were also taken for grafting experiments. Forty-two successful grafts were

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obtained, seven from each source plant. The grafted plants were held in the greenhouse for observation. These plants produced a total of 191 fruits. All were cut in cross section when red ripe. No internal browning was observed in any of the fruits harvested from the grafted plants or the checks.

Two weeks after the third collection the six source plants were lifted, pruned and transplanted to 10-inch pots in the greenhouse. At this time several cuttings were made from each field plant. The rooted cuttings were transplanted and held for observation and a total of 59 graywall-free fruit were harvested from them.

The transplanted field plants were aging and did not grow well; however, there was some new bloom and 15 fruits were harvested. None of these fruits were affected by internal browning.

Several microscopic examinations of graywall tomato plants taken from South Florida fields have failed to disclose a visible pathogen. Mechanical inoculations, grafting, and cutting experiments, have failed to produce any local or systemic foliage symptoms. A total of 504 fruit, all free of graywall, have been harvested from the tomatoes used in the investigations. These data, especially the graft series, indicate internal browning or graywall of tomato in Florida is not due to a transmissible pathogen. Field observations have failed to disclose any correlation of graywall with climatic factors; nevertheless, in view of the results of the investigations to date, it seems reasonable to believe that graywall of tomato in South Florida is caused by physiological factors.

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EVERGLADES EXPERIMENT STATION, BELLE GLADE, FLORIDA

ONION AND SHALLOT DOWNY MILDEW IN LOUISIANA

E. C. Tims

During the past 20 years onion downy mildew (Peronospora destructor) has developed sporadically in Louisiana. During that period, about every third year the disease caused serious damage to the onion crop. But in the last four years, 1947-50 inclusive, there have been three very destructive epidemics. In fact, mildew has caused such serious losses that some farmers who have grown onions for many years have given up the crop completely. For many years mildew caused little injury to shallots. It was observed occasionally but never developed in epidemic proportions. However, in the past five years the disease has become a major problem for many shallot growers especially along the Mississippi River and Bayou Lafourche sections.

DEVELOPMENT IN 1950: Mildew appeared much earlier than usual again this year. In normal seasons it is usually observed during the first half of March but in 1949 and 1950 the disease was found on shallots and onions in January. About the same time mildew was observed on seed onions at Baton Rouge. It continued to develop until about the first of May. Many onion seed stalks that were lightly infected with mildew at the end of April were covered with the black stalk fungus (Stemphylium botryosum). The Stemphylium kept on developing after the mildew fungus had become inactive and apparently helped to kill many seed stalks.

The losses from mildew on shallots in 1950 were probably the heaviest on record. The disease was quite widespread and destructive in the Bayou Lafourche area. Many fields were a total loss. Mildew appeared in the Angola shallot producing area for the first time in 1950.

Green shallot plants do not need very heavy mildew infection to become unmarketable. A moderate amount of leaf infection on shallots that would be of little importance on onions might ruin the shallots for market. Shallots grown for seed (sets) are especially susceptible to mildew injury. The bulbs from diseased plants are usually small and poorly developed. They also keep poorly in storage.

Mildew was also very destructive on onions this year (1950). The losses have been so severe in the past few years that the production of Creole onions has fallen off greatly. The losses are serious on onions grown for bulbs as well as those raised for seed. The onion seed crop was almost a total failure this year. Mildew appeared in the Eunice area for the first time and caused practically a total loss in a number of onion seed fields.

CONTROL MEASURES: Attempts were made to control onion mildew in Louisiana as early as 1919 (Edgerton, 1), but the results were inconclusive. Beginning in 1943, a series of tests for mildew control was begun in Louisiana with a number of fungicides, (Tims, 2, 3). The tests were conducted on onions being grown for seed as well as those being grown for bulbs. Most of the tests were conducted on farms some distance from Baton Rouge. The following fungicides were used as sprays for one or more seasons: lime sulfur, Zerlate, Bordeaux mixture, Fermate, Puratized, Spergon, Phygon, COCS and tribasic copper sulfate. A resin-soap sticker-spreader was used in the early tests and fairly good coverage was obtained. However, most of the spray materials seemed to be washed off by continued heavy rains.

Most of the fungicides gave unsatisfactory results. Lime sulfur, which had proved to be effective against mildew in California, had little or no effect in our tests. Bordeaux mixture showed some promise on seed onions in one test, but in later tests on bulb onions it was ineffective. Zerlate also caused some reduction in mildew injury in one test, but did not give satisfactory control in any of the tests.

A number of dusts have been used on seed onions in a series of tests. None of them gave satisfactory control of mildew when the conditions were specially favorable for the disease to develop. In 1947 a dust containing mineral oil and metallic copper in Bentonite gave promising results in a small test on seed onions at Baton Rouge. Again in 1949 two dusts gave fairly good control on seed onions at Baton Rouge. They were (1) 7% copper, 3% DDT, 2% mineral oil in Bentonite; and (2) 8% Dithane Z-78, 3% DDT in Attaclay.

During the spring of 1950 attempts were made to control mildew in a small onion seed plot at Baton Rouge. The onions were important breeding stock and a special effort was made to control the disease. Mildew had developed rather extensively before control measures were started on January 25. About 300 severely infected plants were pulled and destroyed before the first dust application was made. Later large numbers of infected leaves were removed. During the period from January 25 to May 1, the copper dust was applied as soon as possible after every rain. A total of 16 applications was made during the period of slightly over three months. The dust used contained 7% metallic copper, 3% DDT and 2% mineral oil in Bentonite. In spite of all the precautions taken to save this plot of onion seed, more than half of it was lost.

CONTROL ON SHALLOT: No definite experiments have been attempted to control mildew on shallot. If protective dusts or sprays were applied to shallots they would have to be continued right up to harvest time. This might result in objectional residues being left on the edible leaves. However, the disease is becoming such a serious problem that control measures should be attempted.

In 1947 several rows of shallots were grown for seed next to an onion seed plot. A very severe epidemic of mildew developed in the seed onions and spread to the shallots. In connection with a mildew control test on the onions two rows of shallots were dusted several times with a copper compound containing 6% metallic copper and 1 1/2% mineral oil. Although the disease was well established on the shallots before dusting was started, plants in the dusted rows remained much greener and in better condition than those in the untreated rows.

BREEDING FOR RESISTANCE TO MILDEW: The final solution to the onion mildew problem in Louisiana seems to be the development of resistant varieties. This work has been started at the Louisiana Agricultural Experiment Station in collaboration with the United States Department of Agriculture (4). The Calred variety is quite resistant to mildew in the seed stalk stage and is moderately resistant to leaf infection. This variety has been crossed with the Creole onion at Beltsville, Maryland and the progenies are being tested for resistance to mildew under field conditions in Louisiana.

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DEPARTMENT OF BOTANY, LOUISIANA AGRICULTURAL EXPERIMENT STATION, BATON ROUGE

DISSEMINATION OF ANGULAR LEAF SPOT OF CUCUMBER

F. S. Beecher and S. P. Doolittle

Field observations of the past three years indicate that the organism causing angular leaf spot of cucumber (Pseudomonas lachrymans (E.F.Sm. and Bryan) Carsner) may be disseminated by agencies other than splashing rain, surface drainage water, and cultivation or handling of plants.

In a planting made in 1949 on land that had not grown cucurbit crops for many years, there was no disease until mid-July. At this time plants on the lower portion of the sloping field were inoculated with Pseudomonas lachrymans. After the first symptoms appeared in the inoculated plants it was noted that in some instances the first of the secondary infections were found, not on leaves directly adjacent to those inoculated, but at points two, three, or four leaves removed from the point of original infection. Later it was found that the disease had spread to plants considerably higher up the slope including one hill at the extreme upper end of the field, 100 feet from any other infected plants. In these instances there was no possibility that the bacteria had been carried to the plants by surface drainage water and there had been no handling or cultivation of the plants after the original inoculations were made. The age of the plants at the time the disease appeared made it very unlikely that the infection came from the seed.

In 1950, seed treated with mercuric chloride 1:2000 for 5 minutes was planted in a field where the disease had occurred in the previous year, to test the possibility of overwintering of the organism in the soil. Angular leaf spot appeared before the plants were thinned, and developed first in portions of the field where the disease had not occurred in 1949 and where there was little likelihood that the organism had been carried to the soil by drainage water or cultivation. However, at the time the disease appeared in this field it had been present for some time in other plantings in the vicinity. These accumulated observations were so suggestive of an insect vector, possibly the striped cucumber beetle (Acalymma vittata F.) or the spotted cucumber beetle (Diabrotica undecimpunctata howardi Barber), that an attempt was made to secure more definite evidence as to insect transmission.

On July 19, cucumber seed grown in the greenhouse and known to be free of contamination by Pseudomonas lachrymans was planted on land that had not grown vine crops for at least eighteen years. The rows were spaced nine feet apart, with hills two feet apart, and the seedlings thinned to four plants per hill. In thinning the hands were dipped in mercuric chloride solution before handling each hill, and there was no further handling of the plants. The rows were carefully hand-hoed once without touching the plants, and the cultivator was not allowed to come within two feet of the rows. On August 20 angular leaf spot was present on plants in a number of hills and spread rapidly to other portions of the plot after that date. Bacterial wilt (Erwinia tracheiphila (E.F.Sm.) Holland) also appeared in abundance about the time of the appearance of angular leaf spot.

An older cucumber field, planted during the last few days of May and generally infected with angular leaf spot, lay about 400 yards away on the other side of a small diked stream. This eliminated any possibility of transmission of Pseudomonas lachrymans by drainage water from the old to the new field. The occurrence of angular leaf spot in the late planting, considering previous care in the choice of land and seed, cultivation, and thinning, offers strong circumstantial evidence of dissemination of P. lachrymans by an insect vector. Both the striped and spotted cucumber beetles were present in the plot on August 7 and were very abundant on August 15. The fact that some bacterial wilt appeared in this plot on August 15 indicated that some of

the beetles had come from the older cucumber planting where wilt was prevalent on plants also infected with angular leaf spot.

Some washings from the bodies of spotted beetles, taken in flight in the field where angular leaf spot first occurred, produced infection when sprayed on healthy plants. However, no infection occurred when beetles of either species taken from diseased plants were caged with healthy plants, and so far the evidence of angular leaf spot infection through the agency of these insects remains circumstantial.

DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, BELTSVILLE, MARYLAND

CONTROL OF ANGULAR LEAF SPOT OF CUCUMBER WITH A COPPER FUNGICIDE

F. S. Beecher and S. P. Doolittle

During August 1950 a small test was made to determine the relative efficiency of certain fungicides in the control of downy mildew (Pseudoperonospora cubensis (Berk. and Curt.) Rostow.) on cucumbers. This disease appeared too late in the season to permit any definite conclusions as to the degree of control by the various materials used, but interesting data were obtained on the control of angular leaf spot (Pseudomonas lachrymans (E.F.Sm. and Bryan) Carsner).

The fungicides used were tribasic copper sulfate (3-100), ziram (2-100), and zineb (2-100). They were applied as sprays to plots consisting of 2 rows of 11 hills each. The plots were randomized and replicated three times. The sprays were applied with a small power sprayer using a single hand nozzle. Applications were made at 7-day intervals beginning July 31 and continuing through August 28.

Angular leaf spot had appeared by July 10 and was well distributed over all the plots on August 1. After the first application of the sprays on July 31 the disease continued to spread in the check plots and in those sprayed with ziram and zineb, but in the plots treated with tribasic copper sulfate there was little further infection with angular leaf spot. On August 28 the plants in the check plots showed severe and general injury of the younger foliage by angular leaf spot and the new growth of plants sprayed with ziram and zineb also was badly damaged by the disease. Where tribasic copper sulfate was applied there was almost no spotting of those leaves produced after August 1 and there seemed to be no copper injury to the foliage. Downy mildew and anthracnose (Colletotrichum lagenarium (Pass.) Ell.& Halst.) appeared about August 25, and by September 11 plants in the check plots were dying from the combined effects of these diseases. The plants sprayed with the organic fungicides were in poor condition but those treated with tribasic copper sulfate still were producing some fruit though considerably damaged by downy mildew and some anthracnose infection.

This experiment showed a marked superiority in the control of angular leaf spot by tribasic copper sulfate as compared with ziram or zineb. The organic fungicides did not give adequate control; but after the first application of the copper fungicide there was a surprisingly abrupt check in the spread of the disease.

DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, BELTSVILLE, MARYLAND

STUDIES ON THE PATHOGENICITY OF DIFFERENT ISOLATES OF ASCOCHYTA PISI LIB.

J. D. Gilpatrick and L. V. Busch

Introduction

Leaf and pod spot of peas (Pisum sativum L.) caused by the fungus Ascochyta pisi Lib. is one of the most serious diseases of peas in Canada. It is particularly troublesome in the production of disease-free seed.

There have been very few reports of varietal resistance to this disease. During recent years extensive tests of pea varieties and related species for resistance to A. pisi have been conducted

as an approach to pea improvement in Canada. A certain amount of success has been achieved by the selection and breeding of resistant material (2).

The reports of other workers (1) and the variability in their own results from test to test led the authors to suspect that physiologic specialization occurs in A. pisi. Thus, during 1949 and 1950 the authors collected isolates of A. pisi from several of the major pea growing areas in Canada, and tested their pathogenic capabilities on different varieties of peas and on Vicia villosa Roth.

Source of Isolates -- The different isolates, with their origins, are listed in Table 1. Three of these were obtained from Vicia villosa, upon which A. pisi has been found on several occasions in certain sections of Ontario.

Table 1. Source of isolates of Ascochyta pisi.

Isolate	Location of Collection	Date isolated	Plant Organ	Isolated by
Ottawa	Western Canada	1949	Pea seed	Gilpatrick & Busch
Alberta	Lethbridge, Alberta	1949	Pea foliage	S. G. Fushtey Edmonton, Alta.
Manitoba	Manitoba	1950	Pea seed	E. Robertson District Analyst Winnipeg, Man.
Clinton	Clinton, Ontario	1950	Pea foliage	Gilpatrick & Busch
Vetch #1	Port Dover, Ontario	1949	Vicia villosa, foliage	Gilpatrick & Busch
Vetch #3	Scotland, Ontario	1950	Vicia villosa, foliage	Gilpatrick & Busch
Rosemont	Rosemont, Ontario	1950	Pea foliage	Gilpatrick & Busch

METHODS

All plants used in the tests herein reported were grown in six-inch pots containing steam-sterilized soil. Plants were inoculated four weeks after seeding, or when they had reached a height of approximately six inches depending on the variety.

Inoculation was accomplished by spraying the plants with a spore suspension in 0.02% Aerosol OT[1] solution. This solution was applied to the plants with a hand atomizer or the paint sprayer attachment of a household vacuum cleaner. The Aerosol OT solution dispersed the spores and clung to the foliage, thus producing an effective, even coverage.

Inoculum was prepared by streaking a suspension of spores on pea agar. Abundant sporulation occurred within five days at 20° C. Most cultures were ready for use in a week's time. Medicine bottles of 200 cc. capacity, containing 20 cc. of slanted agar and plugged with cotton were the most satisfactory for culturing. Cultures were carried on pea agar which was found to be a very favorable medium for the sporulation of A. pisi. Potato-dextrose agar induced a mycelial type of development with reduced and retarded sporulation.

Just prior to or immediately following inoculation the plants were placed in a chamber in which a relative humidity of 100 percent was maintained. The temperature with proper ventilation and shading ranged from 65° to 75° F.

The humidity chamber consists of an end of 3/4-inch plywood, 3 feet square, permanently mounted on a bench. This holds the humidifying apparatus. The sides and top are made of light frames covered with cellulose glass. The sections, either 3 or 6 feet long, are held together and to the end with bolts and wing nuts. The chamber is, therefore, capable of being expanded from 3 to 12 feet. The side frames are provided with small doors for the examination and removal of plant material.

At the end of 48 hours the plants were removed from the chamber and placed on the greenhouse bench in the winter or outdoors in the summer. Infection was generally apparent 7 days

[1] A product of the British Drug Company.

after inoculation. At 10 to 14 days after inoculation plants were rated for disease as follows:

0 - No infection
1 - (a trace of infection) 2 or 3 lesions per plant
2 - (slight) 2 or 3 lesions per leaf or stem
3 - (moderate) 4 - 10 lesions per leaf or stem
4 - (severe) 10 to many lesions per leaf or stem
5 - Leaf or stem killed

In establishing the final ratings both the number of leaves infected on an individual plant and the number of plants infected in any one replicate were taken into consideration.

EXPERIMENTATION

The Effect of Time of Planting on Infection

It was felt that the stage of growth of a pea plant at the time of inoculation might be a limiting factor in establishing infection. Thus, the varieties Perfection and Thomas Laxton were each inoculated with the Ottawa isolate at different stages of development, i.e., preflower, early flower and early pod. Sowing seed at bi-weekly intervals provided sufficient material at the proper stages. Appropriate uninoculated checks were also run.

Observations revealed no apparent differences in infection ratings when peas were inoculated at different stages of development i.e., plants from 6 inches high to the early pod stages were all equally susceptible.

The Pathogenicity of Different Isolates

(1) The disease reactions of the Perfection and Thomas Laxton varieties of peas and of V. villosa to 4 isolates of A. pisi were tested. Each variety was inoculated separately by the Ottawa, Alberta, Manitoba, and Vetch #1, isolates. Five plants of each variety were replicated 4 times.

The results, as recorded in Table 2, indicate that there are at least 3 races of A. pisi:

(a) The Ottawa, Manitoba, and Alberta isolates were pathogenic on Perfection and Thomas Laxton, but caused only a trace of infection on V. villosa. The Vetch isolate, however, attacked both pea varieties as well as Vetch.

(b) The Ottawa isolate damaged the leaves and stems of the Perfection variety of peas. The Alberta and Manitoba isolates, on the other hand, while attacking the leaves of this variety, caused little damage to the stems. Thus, if would appear that the Alberta and Manitoba isolates have different pathogenic capabilities from those of the Ottawa isolate, and represent a third race.

Table 2. The reaction of Pisum sativum varieties Perfection and Thomas Laxton, and of Vicia villosa, to four isolates of Ascochyta pisi.

Variety		Degree of infection -- strain: OTTAWA		ALBERTA		MANITOBA		VETCH #1		CHECK	
		Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem
Perfection	Rep. 1	4	1-2	4	0	5	0	5	0	0	0
	2	4	3	4	0	5	1	5	0	0	0
	3	4	2	4	0	5	1	5	0	0	0
	4	3	2	4	2	5	1	5	0	0	0
Thomas Laxton	Rep. 1	3	5	4	5	2-3	5	2-3	1	0	0
	2	3	5	3	5	3	5	2	0	0	0
	3	3	5	3	5	3	5	2	1	0	0
	4	3	5	3	5	-	-	2	0	0	0
Vicia villosa	Rep. 1	0	0	0	0	0	0	2	0	0	0
	2	0	0	0	0	0	0	3-4	5	0	0
	3	1	0	0	0	0	0	2-3	5	0	0
	4	1	0	1	0	0	0	2-3	5	0	0

Table 3. The reaction of six varieties of P. sativum and two varieties of V. villosa to different isolates of A. pisi.

Variety	Degree of Infection by Different Isolates															
	Ottawa		Alberta		Manitoba		Rosemont		Clinton		Vetch #1		Vetch #3		Check	
	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem
Perfection	4	4	2-3	0	4	0	3	4	-	-	2	0	1	0	0	0
Thomas Laxton	2-3	5	3	3-4	3	4	3-4	5	2-3	4	2	0	2	0	0	0
Alaska	3	3	3	2	3	2-3	3	2	3-4	3	1-2	0	2	0	1	0
American Wonder	5	5	-	-	4	4	3	3	3	3	2	0	1	0	0	0
Scotch	2-3	1	1-2	4	2	0	1	0	1	0	1	0	2	0	0	0
O. A. C. 181	2	1	2	2	2	1	3	2	3	2	2	0	0	0	0	0
Chancellor	3	4	2-3	4	4	4	-	-	2	4	2	0	0	0	1	0
A-100[a]	-	-	-	-	0[b]	0	0	0	0	0	1	0	1	0	0	0
Vicia villosa	1	0	1	0	1	0	1	0	2	0	3	2	4	3	0	0
Vicia villosa var. glabrescens	0	0	0	0	0	0	0	0	0	0	2	0	2	1	0	0

[a]Lesions developed slowly. The variety A-100 was obtained from the Division of Horticulture, Central Experimental Farm, Ottawa, Canada.

[b]One plant severely lesioned on leaves and stems.

(2) In the light of the above results, further studies were broadened to include 6 more varieties of peas, V. villosa var. glabrescens, and 3 new isolates of A. pisi. Five plants of each variety were inoculated with each isolate. The results, as recorded in Table 3, confirm the conclusions of the previous experiment. The Vetch #1 isolate differed from the Ottawa, Alberta, and Manitoba isolates with regard to the reaction of Vetch. The Ottawa isolate differed from the Alberta and Manitoba isolates on Perfection.

The Rosemont isolate was similar to the Ottawa, and Vetch #3 was similar to Vetch #1. The exact position of the Clinton isolate was not determined because a reading was unavailable for the reaction of the Perfection variety of peas to this isolate.

The behavior of the Alberta and Manitoba isolates on the Scotch variety suggests that they may be somewhat different. These differences were not confirmed.

The pea varieties O. A. C. 181, Scotch, and A-100 did not appear to be as susceptible to A. pisi as the other varieties tested. These results have been confirmed in field tests. The resistance in A-100 appeared to be a resistance to lesion development rather than to infection. Resistance in O. A. C. 181 and Scotch was to infection, -- lesions, once initiated, developed normally. V. villosa var. glabrescens appeared to be less susceptible to A. pisi than V. villosa.

CONCLUSIONS

Based on the pathogenicity of 7 isolates on 8 varieties of peas, V. villosa and V. villosa var. glabrescens, the results reported here indicate that there are at least 3 physiologic races of A. pisi.

Isolates from peas apparently are only slightly pathogenic on V. villosa, but isolates from V. villosa attack peas as well as Vicia.

The pea varieties O. A. C. 181, Scotch, and A-100 appear to have some resistance to A. pisi.

Since there are different races of A. pisi, it is recommended that in the development of varieties resistant to this organism plant material should be tested against as many isolates of A. pisi differing in pathogenic capabilities as is possible.

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DEPARTMENT OF BOTANY, ONTARIO AGRICULTURAL COLLEGE, GUELPH, ONTARIO

BLACK ROOTROT OF TOBACCO IN ONTARIO AND FACTORS RELATING TO ITS CONTROL [1]

R. H. Stover[2]

Black rootrot of tobacco caused by Thielaviopsis basicola (Berk. & Br.) Ferraris is the most serious disease affecting tobacco in Ontario. The problem of control has been largely solved in the burley tobacco areas with the development and use of the black rootrot resistant varieties Harrow Velvet, Haronova, and Harmony. However, the burley acreage comprises only 10,000 acres, or approximately one-tenth of the total tobacco acreage in Ontario. Thus black rootrot remains a serious problem in the large flue cured area as well as in the relatively small dark tobacco area.

The recently-developed flue cured variety Delcrest is resistant to the disease in the flue cured area and has proved suitable to some but not to all soil types on which flue cured tobacco is grown. There is considerable difficulty in combining black rootrot resistance with certain desirable quality features in flue cured and dark tobacco varieties, and as a result few resistant

[1]Contribution No. 1052, Division of Botany and Plant Pathology, Science Service, Department of Agriculture, Ottawa, Canada.
[2]Junior Plant Pathologist.

lines of these types have been released in Ontario. However, several promising resistant strains of these types are now available and are being further improved in quality at the Harrow Experimental Station.

The problem of breeding for disease resistance is further complicated by the existence of physiological races of T. basicola (6). Certain of the resistant varieties from the southern United States which possess suitable resistance in the areas where they were developed, show little resistance when grown in black rootrot-infested soils at Harrow. However, it has been shown (6) that the fungus in the flue cured area (New Belt) of Ontario is less pathogenic than that in the burley area (Old Belt). The flue cured variety Delcrest possesses 60 percent resistance at Harrow in the Old Belt compared with 80 percent resistance in the New Belt. Therefore, strains of flue cured tobacco developed at Harrow in the Old Belt, with from 50 to 60 percent resistance, will perform satisfactorily in most of the flue cured areas in the New Belt. At present there exists a genuine need not only for several flue cured varieties resistant to black rootrot and adapted to the different Ontario soil types, but also for one or more resistant strains of the dark tobacco type. These must possess quality equal to that of the present widely-grown susceptible varieties White Mammoth and Little Crittenden.

The importance of black rootrot in Ontario may be concluded from an examination of Figure 1 which shows the disease severity in relation to June and July soil temperatures at a depth of four inches for the 10-year period from 1940 to 1950. In 1945, and again in 1950, the disease was very severe and a conservative estimate of loss to the Ontario flue cured tobacco growers in each of these two years would be more than four million dollars. Black rootrot was also severe in 1940, 1941, and 1947. Losses from this disease result not only from a lowering of the crop yield and quality, but also from a general retardation of plant maturity and consequent losses due to early September frosts. Figure 1 indicates a close correlation between low soil temperatures during June and July and incidence of black rootrot of tobacco.

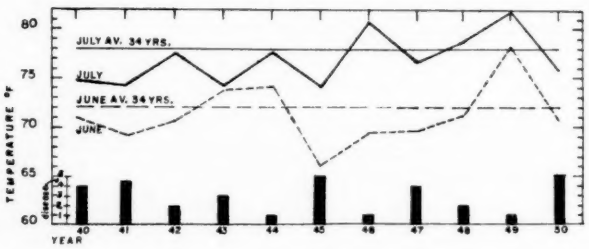

Figure 1. Showing the relation between June and July soil temperatures at Harrow, Ontario and the severity of black rootrot of tobacco. Figures 1 - 5 refer to relative severity of disease as follows: 1 - Very light; 2 - Light; 3 - Moderate; 4 - Severe; 5 - Epiphytotic.

Where resistant varieties are not grown, as is true of most of the flue cured areas and also the dark tobacco areas, rotations are depended upon to control the disease. The standard practice in the flue cured area is a 2-year rotation in which rye alternates with tobacco. On soils where the pH is above 6.0, a 2-year rotation is often insufficient for control of black rootrot. Also, in certain areas where the pH is near 7.0 even a 3-year rotation may be insufficient to control the disease. Generally, in the flue cured area where only rye and tobacco are grown, a 3-year rotation consisting of 2 years of rye and 1 year of tobacco offers a sufficient measure of control. In the dark tobacco area, however a 5-year rotation is considered necessary, and the disease may be severe in certain years in fields where tobacco has not been grown for eight to ten years, or even longer. The reason for this probably lies in the fact that T. basicola not only attacks tobacco but also colonizes legume hosts in Ontario (6). Some of the typical rotations

used in the dark tobacco areas involving legumes are shown in Table 1. In this table it is shown that even where two years of wheat or corn are grown between legume crops the disease may still be present, and that legume crops preceding tobacco may be conducive to injury from black rootrot. Also, because of the ease with which T. basicola is isolated from such soils and from legumes by using potato discs (5) it is evident that T. basicola can maintain itself, and multiply to a certain extent on hosts such as soybeans, beans, red clover, sweet clover, and alfalfa in Ontario. If a susceptible variety of tobacco such as Little Crittenden is planted even every five years in a rotation involving legumes in soil infested with T. basicola, the crop may be attacked, the fungus having maintained itself on the legume crops in the intervening years. Evidence will be presented elsewhere that certain strains of this fungus are adapted to hosts other than tobacco.

Table 1. Crop rotations in relation to the 1950 incidence of black rootrot in soils from the dark tobacco areas of Ontario.

	Rotations							
								Tobacco--1929.....
1942								Beans
1943			Tobacco					Wheat
1944			?					Alfalfa
1945	Corn	Corn	?		Tobacco	Sweet clover		Alfalfa
1946	Corn	Beans	Wheat		?	Wheat		Alfalfa
1947	Beans	Tobacco	Corn	Beans	Wheat	Red clover		Alfalfa
1948	Corn	Wheat	Corn	Wheat	Red clover	Red clover	Tobacco	Beans
1949	Beans	Wheat	Soybeans	Alfalfa	Corn	Red clover	Corn	Wheat
1950	Tobacco	Red clover	Tobacco	Tobacco	Beans	Tobacco	Soybeans	Tobacco
Relative injury to tobacco by T. basicola	Moderate		Moderate+	Severe		Moderate+		Moderate in one area of field
Relative growth on potato discs of T. basicola from soil	+++	++	+++	+++	++	++	++	+

\+ sparse, ++ medium, +++ abundant

It is evident from the above data that in some of the tobacco areas of Ontario (Old Belt), rotations of five or more years which include legumes will not necessarily reduce the infestation of T. basicola. Injury will occur in these areas when susceptible varieties are planted in seasons when soil temperatures are favorable for disease development.

In addition to resistant varieties and crop rotations, preliminary experiments indicate that certain soil fumigants offer some promise for the control of black rootrot of tobacco in Ontario. Koch and Stover (3) have shown that the fumigants dichloropropene-dichloropropane, chloropicrin, and ethylene dibromide are all effective in controlling brown rootrot of tobacco in Ontario. In Table 2 and Figure 2 the results of fumigation tests involving soil infested with the black rootrot fungus are given. These tests were carried out at temperatures of 68° F. using naturally-infested black rootrot soil (pH 6.6) that had grown tobacco for eight consecutive years. Three ml. of fumigant was placed in the centre of each four-inch pot (10 pots per treatment). After 17 days the pots were seeded with the burley varieties Green Briar (susceptible) and Harrow Velvet

(resistant). One month later the average green weight of five plants from each treatment was recorded and these are listed in Table 2.

Table 2. The effect of soil fumigants on the weight of tobacco plants growing in soils infested with the black rootrot fungus.

Treatment	Variety	
	Green Briar	Harrow velvet
	Wt. in gm.	
Dowfume N	7.0	8.7
Dowfume H-940*	1.6	3.0
Dowfume G	.8	1.7
Check (no treatment)	.7	3.7

*26% by weight of methyl bromide and 74% xylene

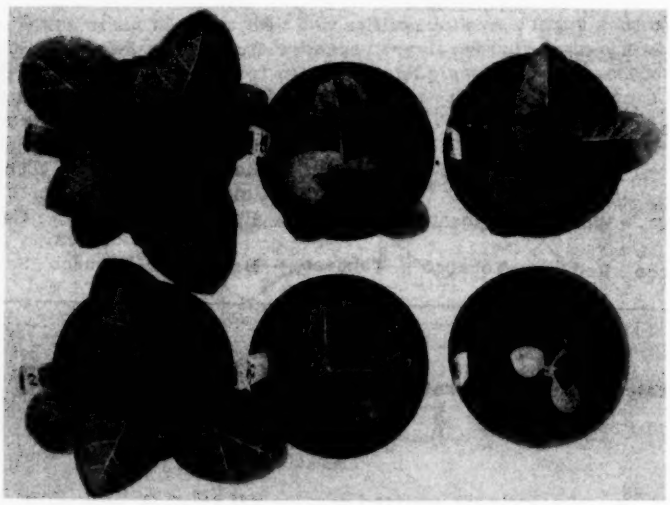

Figure 2. The effect of soil fumigants on the growth of tobacco in soils heavily infested with black rootrot. Upper row variety Harrow Velvet: left to right, fumigation with Dowfume N; H-940; check, no treatment. Lower row variety Green Briar: left to right, fumigation with Dowfume N; H-940; check, no treatment. Note that Green Briar is susceptible to and Harrow Velvet is resistant to black rootrot. Note also the increased growth of both varieties where the soil was fumigated with Dowfume N.

Table 2 indicates that Dowfume N (dichloropropene-dichloropropane) is highly effective in controlling the disease under these experimental conditions. The increased growth of the variety Harrow Velvet, which is highly resistant to black rootrot but completely susceptible to brown rootrot, indicates that Dowfume N may have acted as a nematicide as well as a fungicide in this experiment.

The fungicidal action of volatile soil fumigants towards T. basicola has been reported by Zentmeyer and Kendrick (7) who found that Dowfume N killed this fungus under sterile conditions in closed jars. In North Carolina, Lucas (4) found that dichloropropene-dichloropropane applied in 9-inch furrows did not control black rootrot. Various chemicals added to the soil have been reported as effective in reducing the incidence of this disease (1, 2). Experiments in Ontario indicate that a fumigant such as dichloropropene-dichloropropane that offers possible control for

both black and brown rootrot merits further testing. Therefore, it is planned to continue these experiments under field conditions in 1951. It would seem not unlikely that one or more soil fumigants may be found to be effective in controlling black rootrot of tobacco, and that such fumigants may prove practical for use in certain heavily-infested areas for which resistant varieties are not yet available.

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LABORATORY OF PLANT PATHOLOGY, HARROW, ONTARIO, CANADA

NOTES ON DISEASES OF SOME SPECIAL CROPS IN 1950

C. A. Thomas

SAFFLOWER: Rot root (Phytophthora drechsleri) was very destructive in southern California in the late spring. Large plantings were made in that area with varieties N852 and N9, which are the most susceptible varieties known. Over-irrigation and "long runs" and certain other cultural practices were contributing factors. Root rot also was observed in Arizona, Oklahoma, Texas, Kansas, Colorado, and Nebraska. In the latter State the disease was less serious this year than in the past two seasons, owing to the fact that susceptible varieties such as N852 and Indian were planted mainly on dry land and resistant varieties such as N8 were grown under irrigation.

Rust (Puccinia carthami) was much more prevalent this year in Colorado and Nebraska than in 1949. This was thought to be due to a build-up of inoculum in the area. Certain growers who failed to rotate safflower experienced a loss in stand from rust infection in the seedling stage. A few safflower plantings affected with rust were observed in Kansas and California.

Leaf spot (Alternaria carthami) was of minor importance in Colorado and western Nebraska, but was prevalent in experimental plantings in central and eastern Nebraska, Kansas, Oklahoma, and Texas under more humid conditions. None was observed in Arizona or California. In the latter two States certain unidentified species of Alternaria and Botrytis caused some damage to leaves and buds.

CASTOR BEAN: Unusually wet summer weather in Oklahoma and northern Texas resulted in considerable capsule mold (Alternaria ricini). Capsules became infected early and the seed failed to develop completely. Capsule mold (Botrytis and Monilia spp.) caused a limited amount of loss in certain sections of California.

Plants in an experimental planting at Shafter, California were affected with what was tentatively identified as Verticillium wilt. During July, affected plants exhibited yellowing of the leaves. Leaves cupped upward and finally dried and abscised at the base of the petiole. Affected plants were stunted and wilted at lower temperatures than normal plants. Capsules that were filling at this time dried up and produced light or hollow beans. In early August many of the plants began making new growth. All varieties in the planting were affected. The soil is known to be heavily infested with Verticillium albo-atrum which causes wilt in cotton.

MINT: Verticillium wilt (V. albo-atrum var. menthae Nelson) was unusually severe on peppermint in Indiana and Michigan. Spearmint was also affected. Weather conditions were particularly favorable for heavy infection and development of the disease.

Rust (Puccinia menthae) caused considerable damage to spearmint in the Midwest and many growers cut the herb early to avoid losses resulting from leaf fall.

Reports from Oregon indicated that peppermint was severely damaged by rust in that State. Although rust was prevalent on spearmint in the Midwest, none was observed on peppermint.

At Beltsville, Maryland, observations on rust and anthracnose (Sphaceloma menthae) showed that M. spicata (spearmint), M. longifolia, M. arvensis var. piperascens (Japanese mint), M. rotundifolia, and M. sylvestris were affected with rust but not with anthracnose. M. piperita (peppermint), M. canadensis, and M. citrata were affected with anthracnose but not with rust.

SESAME: The development of a commercial acreage of sesame in this country is dependent upon the production of high yielding, indehiscent types by the plant breeders. Definite progress has been made in the development of such types, and these, along with the best dehiscent varieties, are being widely tested in the Southern States. Experimental plantings have been examined to determine which diseases may be serious in the event of large scale cultivation of the crop.

Cercospora sesami has been observed in Maryland, South Carolina, and Texas. The organism is known to be seed-borne and sometimes causes early defoliation of some varieties.

A bacterial leaf spot organism, tentatively identified as Pseudomonas sesami, appears to cause one of the most serious diseases of sesame. The trouble has been observed in plantings from Maryland to Texas. At College Station and Chillicothe, Texas the disease was very severe during the past season. Most varieties appear susceptible and certain introductions from Turkey are severely damaged. A few late maturing types appear to have some resistance. Late plantings at Chillicothe were a complete loss from this disease.

A leaf spot caused by Alternaria sp. was very prevalent at Knoxville, Tennessee. Fusarium wilt has been observed in certain sections of the Southeast.

DIVISION OF TOBACCO, MEDICINAL, AND SPECIAL CROPS

SMALL GRAIN DISEASES IN NORTH CAROLINA IN 1949-50

J. G. Moseman and T. T. Hebert

Most of the small grain produced in North Carolina is sown in small fields in the fall. According to the North Carolina Department of Agriculture, Division of Agricultural Statistics, the number of acres of each of the grains harvested and their yields in 1950 was as follows: barley, 37,000 acres, averaging 25 bushels per acre; wheat, 423,000 acres, averaging 14.5 bushels per acre; oats, 388,000 acres, averaging 30 bushels per acre.

Observations of the diseases in commercial small grain fields were made throughout the State. Detailed notes were also taken on the nurseries at McCullers in Central North Carolina, and at Statesville in the Piedmont region. Periodic inspections were made in the nurseries at Plymouth on the Coastal Plains, and at Waynesville in the Western Mountain region.

BARLEY: Leaf rust (Puccinia hordei), which was present over the entire State, reduced the yield of barley an average of about 10 percent. One 20-acre field of Sunrise, near Cary, was so heavily infected that it was not worth harvesting. An adjoining 20-acre field of Wong, which is somewhat more tolerant of rust, was not killed so early but it likewise was not worth harvesting.

There was very little stem rust (Puccinia graminis) on barley. It developed on some of the later varieties, but did not cause much damage.

Powdery mildew (Erysiphe graminis) was very prevalent in the fall but was not so severe in the spring. The inoculum from the fall infection should have been ample for spring infection, but the weather conditions were not favorable for its development this spring. It is estimated the average yield for the State was reduced about 5 percent by this organism owing primarily to the heavy fall infection.

There was from a trace to 10 percent of loose smut (Ustilago nuda) present in most fields throughout the State.

The losses from Helminthosporium spp. were not so great as in previous years, and caused only about a 2 percent reduction in the average yield. Barley stripe (H. gramineum) was found in the nurseries at Waynesville, Statesville, and McCullers; it did not cause an appreciable amount of damage at any of these places. Spot blotch (H. sativum) was present in many fields, but severe damage occurred in only a few of them.

Scab (Fusarium spp. and Gibberella zeae) was so severe on some varieties in the nursery at Statesville that they produced very little grain. Varieties appeared to vary in their degree of susceptibility. An Iredell-Bolivia selection, which was resistant to mildew and rust, but appeared to be very susceptible to scab, ranked 48th in yield out of 49 varieties tested in the Advanced Barley Yield Nursery at Statesville. The loss caused by this organism is hard to estimate but it probably amounted to an average of about 5 percent.

Barley scald (Rhynchosporium secalis) was first found in the North Carolina Piedmont section in the spring of 1946. Only two infected fields were found that year. In 1947 and 1948 the number of infected fields observed increased, and in 1949 scald was the most important disease of barley in the western part of the State. Practically every field visited was fairly heavily infected and many plants were severely defoliated by the disease. Scald was less severe in 1950, but was found in every section of the State. The average loss is estimated at 2 percent.

No covered smut (Ustilago hordei) was observed this year.

WHEAT: Leaf rust (Puccinia rubigo-vera tritici) was prevalent and caused considerable damage since only a small proportion of the acreage was planted to resistant varieties. It is estimated that the yield of wheat was reduced 10 percent by this organism.

Powdery mildew (Erysiphe graminis) caused about the same amount of damage as did leaf rust. It was prevalent in the fields over the entire State, both in the fall and spring. Apparently the weather conditions were more favorable this spring for the development of mildew on wheat than on barley.

Loose smut (Ustilago tritici) was observed in most of the fields visited. The average losses caused by this disease did not amount to more than 1 or 2 percent.

Glume blotch (Septoria nodorum) was very prevalent this season. It caused spots on the leaves and blackening of stems, as well as the usual head and node lesions. It was found throughout the State but was most severe in the Piedmont region, and possibly reduced the yield an average of about 2 to 5 percent. The variety Purcam seemed very susceptible to this disease.

Speckled leaf blotch (Septoria tritici) was observed only in the Wheat Mosaic Nursery at Statesville, where the lower leaves of some of the varieties were killed by this organism.

Wheat mosaic was reported in two fields of Atlas 66 wheat this year. One of these fields was located about 35 miles Northwest of Raleigh, and the other near Salisbury. Only one or two acres in each field were affected. There was very little mosaic in the Wheat Mosaic Nursery at Statesville.

Scab (Fusarium spp. and Gibberella zeae) was observed on wheat in the nursery at Statesville. Because of the large amount of S. nodorum present it was difficult to evaluate the damage caused by this organism, but it probably did not exceed an average of 2 percent.

Twist disease (Dilophospora alopecuri) was first found in North Carolina in a field near Kings Mountain in the spring of 1948. A loss of 5 percent was estimated for this field. It was present in a few fields in the southern Piedmont section of the State in 1949 and again in 1950. This disease was always found associated with the wheat nematode, Anguina tritici. Losses from both of these diseases were minor.

No bunt (Tilletia spp.) or stem rust (Puccinia graminis) was observed in the State this past season.

The varieties Redhart, Coastal, and Purplestraw suffered greatly from frost damage at McCullers and Statesville.

OATS: Crown rust (Puccinia coronata) was very prevalent in the fall in eastern North Carolina. Some fields were so badly damaged that the oats were plowed under. This spring crown rust infection was exceedingly light. However, the severity of the fall infection may have reduced the average yield about 2 percent.

Stem rust (Puccinia graminis) was found in only one or two fields, but caused no appreciable damage.

Powdery mildew (Erysiphe graminis) on oats has increased in the State in the past few years. There was sufficient mildew present in the nursery at Statesville to kill the lower leaves on some varieties. There appeared to be differences in the susceptibility of the varieties.

The smuts (Ustilago avenae and U. kolleri) were not very prevalent in any of the nurseries. However, some selections from crosses involving Santa Fe were heavily infected. The loss due

to these two organisms amounted to an average of about 2 percent.

Spot blotch, Helminthosporium sativum, was isolated from plants collected from several fields in the fall, but the loss amounted to only about 2 percent. The variety Southland, C. I. 5207, was severely damaged by this organism at Plymouth.

Leaf blotch, Helminthosporium avenae was the most serious disease on oats this past season. It was observed in fields throughout the State. In many fields practically all the leaves on the plants were infected. The amount of damage contributed by this organism is hard to estimate, but it probably exceeded an average of 10 percent reduction in yield of grain.

Victoria blight of oats (Helminthosporium victoriae) was first observed in North Carolina in the spring of 1949. It was found in Hyde County in the eastern part of the State where it was present in only a few fields. Estimated yield losses in these fields in 1949 ranged from 10 to 50 percent. A survey of this area in 1950 showed that the disease was still present but was not so severe as in the previous season. It was also found in another county, Onslow, in the eastern section of the State this year.

Mosaic of oats was found in some fields throughout the Piedmont and Coastal Plains sections of the State, but since most of the varieties grown commercially are somewhat tolerant to the virus, damage probably did not exceed 1 percent.

Halo blight (Pseudomonas coronafaciens) was observed in two commercial fields south of Plymouth. It was not observed in any other part of the State.

Fields throughout the State had scattered areas of plants with reddened leaves early in the spring. There was no indication that this discoloration was due to a pathogen. Isolations were made from leaves and roots of affected plants from several locations, but no pathogen could be found consistently. There were some indications that the condition was exaggerated by cold temperatures. It may also have been influenced some by the fertility level of the field. There appeared to be differences in varietal response. Observations made in the nursery at McCullers on April 14 showed that the plants had begun to recover from the pigmentation disorder. The new leaves of plants which earlier had showed the discoloration were then normal green in color.

DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND NORTH CAROLINA AGRICULTURAL EXPERIMENT STATION, RALEIGH, NORTH CAROLINA

OBSERVATIONS ON CORN DISEASES IN VIRGINIA FROM 1947 to 1950

Curtis W. Roane[1]

The most important widespread diseases of corn in Virginia are stalk rot, ear rot, leaf blight, and common smut. Each year, these diseases cause heavy losses to corn growers in Virginia. Most growers have been acquainted with smut for many years and do not become alarmed at its appearance. The introduction of hybrid corn into Virginia has brought an increasing number of inquiries about stalk rot, ear rot, and leaf blight.

The most striking disease in 1947 and 1948 was leaf blight, caused by Helminthosporium turcicum. It caused considerable damage to U. S. 13 and U. S. 99. A number of other commercial hybrids proved to be highly susceptible. The disease caused by this organism was not very severe in 1949 and 1950, but in 1949 it was accompanied by Cochliobolus heterostrophus (H. maydis) which is usually of little importance, but which in 1949 completely blighted leaves of commercial hybrids in southeastern Virginia. C. heterostrophus spread over the entire State that year; but in 1950, it moderated and occurred only in the southeastern quarter. Even there, damage was not so great as in 1949. Its occurrence at Blacksburg in our breeding nurseries in 1949 confounded our blight resistance progress. Many previously isolated lines resistant to H. turcicum were completely susceptible to C. heterostrophus. A number of experimental hybrids proved to be resistant to both of these organisms.

The perfect stage of C. heterostrophus was found on a number of plants in the Holland area. It usually occurred on or near the collar. The asci were mature by early September.

[1]The writer is indebted to S. B. Fenne, Pathologist, Agricultural Extension Service, and C. F. Genter, Agronomist, Agricultural Experiment Station, for directing his attention to a number of the diseases cited.

In 1949 and 1950, stalk rot, caused primarily by Gibberella fujikuroi and frequently by Diplodia zeae, was the most important disease in Virginia. Root rot usually accompanied this disease. In 1949, severe breakage of hybrid U. S. 357 occurred in eastern Virginia. At the end of the season broken stalks were covered with a pink mold which proved to be Fusarium moniliforme. The perithecial stage, G. fujikuroi, was abundant. In 1950, U. S. 262 and U. S. 357, both recommended hybrids, broke severely. In some fields, 100 percent of the plants were infected and 75 to 90 percent had fallen by September 8. These varieties were not dry enough to be stored safely until October 1, so that breakage approached 100 percent by harvest time. At Holland, the progress of the disease was observed at intervals and an account is given here for hybrid U. S. 357.

By August 2, numerous purple lesions had appeared on the leaf sheaths. Sporulation occurred only where the Gibberella infections coincided with brown spot lesions (caused by Physoderma zeae-maydis), the latter being very common. No internal necrosis occurred at that time and only rarely was the outer cortex infected. P. zeae-maydis attacked only the leaf blade and sheath, the culm being infected only rarely at the node and therefore seldom affording an avenue of entrance to the culm for G. fujikuroi. By September 5, G. fujikuroi had entered the stalks, rotted the nodal sclerenchyma, and much of the pith. Breakage had occurred up to 90 percent. Examination of plants with the least rot revealed that the fungus apparently entered the node from the infected leaf sheath. The rotting started as a wet rot in the nodes and progressed until the pith was soggy. After a period of time, the nodal sclerenchyma and pith dried up, leaving only the vascular system within the cortex. The cortex was progressively weakened, particularly at the nodes, until the stalk broke. Pure cultures of G. fujikuroi could be isolated during all stages of the disease.

The pedigrees of U. S. 262 and U. S. 357 are respectively (Kys x Hy) x (C.I. 2 x C.I. 3) and (Kys x C.I. 7) x (C.I. 3). It is significant that both have the same pollen parent and one inbred common to the seed parent. U. S. 99 and U. S. 265, which also have C.I. 2 x C.I. 3 as their pollinator, have broken severely in the past.

Other hybrids, which rotted less than U. S. 262 and 357, were found to harbor Gibberella fujikuroi and Diplodia zeae. Isolations from them yielded equal numbers of Gibberella fujikuroi and Diplodia colonies, but rarely were the two fungi isolated from the same node. G. zeae and Nigrospora oryzae were isolated from several stalks in conjunction with the breakage described above.

In 1948 and 1949, isolated cases of stalk rot caused by Pythium butleri were reported from Northampton and Nelson Counties, respectively. Specimens from Northampton were collected by T. J. Nugent, Virginia Truck Experiment Station, and identified at the Division of Mycology and Disease Survey. U. S. 13 was the Nelson County victim and the outbreak occurred on river bottom land.

All the organisms causing stalk rot, except Pythium butleri, have been found to cause root rot and ear rot. In addition, a blue Penicillium sp., Helminthosporium carbonum, Botrytis cinerea, Aspergillus niger, Cephalosporium acremonium, and Cunninghamella sp. have been isolated from ears showing various degrees of rot. The Gibberella spp., Diplodia zeae, and Nigrospora oryzae caused the major losses.

Gray leaf spot, caused by Cercospora zeae-maydis[2] was first observed at Blacksburg in 1949. It appeared again at Blacksburg in mid-August of 1950 and became very severe by September 1. Various degrees of resistance were observed for inbred lines, but double crosses involving 100 experimental hybrids expressed no resistance. Complete destruction of the leaves resulted. Many single cross combinations were observed and those having C.I. 21E or M 14 as parents were susceptible to the disease. The disease was also found on popcorn and sweet corn at Blacksburg in 1950 and on field corn in Roanoke and Tazewell Counties at elevations of 1600 and 3300 feet respectively. In general, gray leaf spot is an unimportant disease of corn, but when it appears early in the season, severe damage may result. It was conspicuous in 1950 because Helminthosporium turcicum did not appear in time to destroy the leaves ahead of it.

Corn rust, caused by Puccinia sorghi, has occurred each year since these observations were begun. In 1950, it caused concern among the growers. Like Cercospora zeae-maydis, it was conspicuous in the absence of Helminthosporium turcicum whose effects usually overshadow other folicolous corn parasites. On late-planted sweet corn in the Blacksburg area, rust defoliated plants below their ears and damaged the upper leaves. H. turcicum blight destroyed the remaining foliage. The two diseases caused about 60 percent loss of June- and July-planted 80- to 90-day sweet-corn varieties in this area.

Bacterial wilt (Bacterium stewarti) has occurred each year during this study on susceptible varieties of sweet corn; and in 1950, it was severe in Montgomery County. Flea-beetle damage

[2]Identified by J. A. Stevenson, Division of Mycology and Disease Survey.

was much greater than in the three-year period and could possibly be correlated with wilt severity.

In 1950, anthracnose (Colletotrichum graminicola) accompanied early rust infection on sweet corn and accounted for considerable damage to the first three leaves in several gardens. In combination with rust, it retarded early plant development.

Corn diseases were destructive in 1950 because of weather conditions favorable for their development and because of the general use of highly susceptible hybrids. Rainfall was abundant and humidity was high throughout most of the State until August. Conditions were especially favorable for stalk rot development and infection occurred early and progressed rapidly during the warm dry August. It is estimated that stalk breakage and subsequent ear rots will cost Virginia farmers 15 to 20 percent of their crop this season.

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CERATOSTOMELLA PARADOXA DADE ON BANANA

G. B. Ramsey and M. A. Smith

Thielaviopsis paradoxa is well known as a parasite of such tropical plants as pineapple, sugar cane, coconut palm, oil palm, and banana. Up to the present time, however, apparently the perfect stage has been found only on discarded cacao husks. In 1928 Dade (1) described the perithecial stage of this fungus on cacao husks and designated it as Ceratostomella paradoxa Dade (= Endoconidiophora paradoxa (de Seynes) Davidson). The present note is to report the perfect stage on banana stalks from Central America.

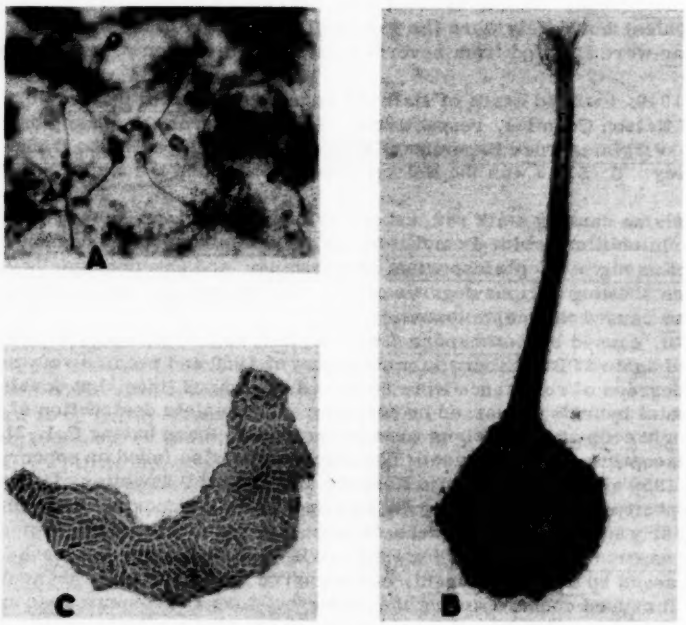

Figure 1. Ceratostomella paradoxa on banana.
A Perithecia on cut stalk of banana.
B Perithecium showing fimbriate beak with a mass of ascospores.
C Mass of ascospores in a fatty matrix lifted from the beak of a perithecium.

While studying the market diseases of bananas in Chicago, January 1950, the perfect stage was found in great abundance on the tips and butts of banana stalks after the fruit had been ripened. Green fruit and stalks on arrival at the market and ripening rooms frequently show slight to moderate development of the Thielaviopsis stage at the cut tips and butts of the stalks, but usually it is not until the bunches (stems) have been in the ripening room for a few days that the perfect stage becomes evident. Most of the perithecia are developed on the tough, fibrous stalk and cushion tissues where the cuts were made at harvesting time or where splits have occurred (Fig. 1, A, B). On this fairly dry, fibrous material the black perithecia are conspicuous because they are mostly superficial and the long beaks with the globular spore mass on the fimbriate tip of each make them visible to the naked eye (Fig. 1, B). These beaks are 800-1000 microns long and 35-40 microns in diameter. The bases of the perithecia usually range between 250-and 300 microns in diameter. The ascospores measure 2.6-4.1 x 7.2-10.4 microns, averaging 3.2 x 8.5 microns (Fig. 1, C).

Perithecia have developed in a few instances in a culture medium composed of a decoction of banana stalk to which dextrose and agar were added. As a rule, however, isolations from banana stalks and fruit develop only the Thielaviopsis stage on this medium as well as on potato-dextrose agar. Of the numerous cultures studied none has ever produced perithecia on potato-dextrose agar.

The measurements of the spores and perithecia of the banana isolates correspond closely with those of the fungus obtained by Dade from cacao husks. The causal organism of banana black rot of the fruit and of banana stalk rot should therefore be referred to Ceratostomella paradoxa Dade.

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DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE; CHICAGO, ILLINOIS

A SURVEY OF DISEASES LETHAL TO TAHITI (PERSIAN) LIMES IN DADE COUNTY, FLORIDA[1]

C. M. Gates and M. J. Soule, Jr.

In recent years considerable concern has been felt by growers over the failure of groves of the common Tahiti and the Idemor varieties of limes. Many of the groves in the Redlands District of Dade County have had as high as 50 percent of the trees die out in the last few years.

In 1934 Tisdale (2) described a bark disease of the Tahiti lime caused by Diplodia natalensis Pole-Evans, and Diaporthe citri Wolf. In 1943 Ruehle (1) described the symptoms of a disease believed to have a virus as its causal agent and which was thought to be associated with the Diplodia lime bark disease.

The present survey was made to determine the amounts of lime bark disease and presumed virus disease present in Dade County and to attempt to correlate the presence of the virus disease to the severity of the Diplodia and Diaporthe bark rots.

From 40 to 50 trees in 18 groves over three years old were surveyed and sampled. The trees were checked for lesions of lime bark disease on the trunk and larger branches and the fruit and foliage was inspected for the presence of virus symptoms as described by Ruehle.

A summary of the data collected in this survey is given in Table 1. Of a total of 844 trees in 18 groves 38.5 percent had trunk lesions and 31.5 percent showed virus symptoms. No groves in this group were completely free of lime bark disease, from 8 percent to 78 percent of the trees being diseased. Only three groves were free of virus symptoms, and in one grove 94 percent of the trees showed the typical streaked fruit and mottled leaf chlorosis. The prevalence of lime bark disease increased sharply with the age of the groves up to 15 years. The percentage of virus symptoms decreased in groves over 10 years old.

[1]This is a summary of a paper which will be published in the 1950 proceedings of the Florida State Horticultural Society.

Table 1. A summary of data collected in a survey of Dade County lime groves to determine the amounts of lime-bark and virus disease present.

Grove	Variety	Root stock	No. of trees	Total trunk lesions %	Total virus symptom %	High bud lesions %	Low bud lesions %	Virus with lesions %	No virus with lesions %
				Groves less than 3 years old					
A	I[1]	RL[2]	50	0.0	0.0	-	-	-	-
B	I	RL	50	0.0	0.0	-	-	-	-
C	T[3]	RL	50	0.0	2.0	0.0	0.0	0.0	0.0
	Totals		150	0.0	0.6	0.0	0.0	0.0	0.0
				Groves 3-6 years old					
D	I	Cleo[4]	44	34.1	91.1	21.1	48.0	40.5	0.0
E	T	RL	50	12.0	6.0	10.3	18.2	33.3	10.6
F	T	RL	50	14.0	72.0	15.2	0.0	16.7	7.1
G	T	RL	50	8.0	0.0	0.0	8.7	-	8.0
H	T	RL	50	8.0	60.0	3.8	12.5	3.3	15.0
	Totals		244	14.8	44.6	11.9	19.1	22.0	9.6
				Groves 6-10 years old					
I	I	RL	42	42.9	42.9	21.4	57.1	55.6	38.1
J	T	RL	50	18.0	0.0	12.5	19.0	-	18.0
K	T	RL	44	54.5	54.5	45.0	62.5	75.0	25.0
L	T & I	RL	50	44.0	94.0	43.8	44.4	40.4	100.0
	Totals		186	44.6	47.8	36.5	42.0	52.8	26.6
				Groves 10-15 years old					
M	T & I	RL	50	36.0	36.0	36.0	-	56.3	28.1
N	T	RL	50	76.0	8.0	81.4	42.9	75.0	76.1
O	T	RL	50	40.0	8.0	38.8	100.0	75.0	37.0
P	T	RL	39	69.2	20.5	60.7	90.9	100.0	45.0
Q	T	GF[5]	50	78.0	42.0	78.0	-	81.0	72.4
	Totals		239	59.4	23.0	58.2	73.7	77.4	51.8
				Groves over 15 years old					
R	T	RL	50	16.0	4.0	15.9	16.0	100.0	12.5
S	T	RL	50	14.0	18.0	15.6	0.0	55.6	7.3
T	T	RL	50	60.0	4.0	71.8	45.0	100.0	72.0
U	T	GF	25	72.0	0.0	72.0	-	-	58.3
	Totals		175	36.0	7.4	32.7	27.3	69.2	34.0
	Grand total		994	31.6	26.9	33.1	29.6	45.5	26.4
Total less A, B, & C			844	38.5	31.5	39.8	33.5	45.7	33.1

[1] I- Idmore
[2] RL- Rough lemon
[3] T- Tahiti
[4] Cleo- Cleopatra mandarin
[5] GF- Grapefruit

The percentage of trees with no virus symptoms but with trunk lesions increased with age at about the same rate as the percentage of total lesions. The percentage of trees with virus symptoms that had trunk lesions increased more rapidly and was consistently higher. This indicates that the presumed virus disease is probably a contributing factor to the prevalence of lime bark disease.

The height of the bud union seemed to be a more important factor in lime bark disease in trees under 10 years old. A tree was considered to have a low bud if the bud union was at or below the

soil surface. In the younger groves the low-budded trees consistently had more trunk lesions than the high-budded trees. In some of the older groves there was as much or more bark disease among the trees with high buds.

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UNIVERSITY OF MIAMI, TROPICAL PLANT AND FOOD RESEARCH, CORAL GABLES, FLORIDA

DRY WEATHER AND TREE TROUBLES IN MASSACHUSETTS [1]

David H. Marsden[2]

During the past few years these Laboratories have noticed a marked increase in the number of troubles, and complaints and inquiries concerning troubles, of trees and shrubs which were attributed primarily to a lack of soil moisture. These maladies include needle blight of conifers, early leaf-fall of deciduous trees, die-back or small, thin foliage on established trees, transplanting failures, widespread sun-scorch of maples, outright dying of evergreens over appreciable areas and the accelerated dying of oaks in areas previously ravaged by the Gypsy moth.

By blaming the "weather" in such instances we have perhaps not always satisfied the owners of the afflicted trees, some of whom are inclined to think that this is an answer designed to avoid further investigation or to hide one's ignorance. Especially is this opinion likely to be formed where trees are concerned, since trees and shrubs, unlike herbaceous plants, are not so quick to react visibly to periods of dry weather. Consequently, the symptoms of ill-health and the diagnosis of drought injury may come at a time when there is adequate soil moisture, and lawns, flowering annuals, and garden crops are doing well.

However, it is not so much to justify the position of the plant pathologist in such instances that the writer has undertaken this study, as to analyze in some detail the pattern of precipitation over the past several years and the possible effects on tree growth and health.

In Massachusetts, at least, the period 1939 through 1949 has been decidedly dry, with only two years (1942, 1945) having a greater than average precipitation. These eleven years have resulted in a total deficit of 35.77 inches which is an annual average deficit of 3.25 inches as based on the average annual precipitation of 43.70 inches.[3] These figures apply for western Massachusetts. The eastern half of the State receives less average precipitation (40.14 inches) and has experienced more severe droughts in recent years. The year 1950 also promises to be a dryer than average one. This lack of "normal rainfall" with several drought or near-drought periods over the indicated period has been apparent to most of the public who have seen their lawns and gardens wither and have been urged at times by civic authorities to curtail the use of water during the worst of the dry spells. A below average annual rainfall is not in itself the whole story, however.

There is no such thing as a "normal annual precipitation". There is rather a fluctuation from one year to the next which can be averaged over an extended period of years to give a figure which is called normal for a given area. For this reason, the figure given as the normal precipitation, while a helpful concept in many respects, may be misleading where differences in the supply of moisture to perennial plants are being considered. In order to see more correctly the actual severity of a dry period with its possible consequences to woody plants it would seem to be better to compare the volume of precipitation during this period with that of a period of the same length preceding or following it. Furthermore, since the effect of dry weather on trees and shrubs is more serious during certain parts of a year, it is desirable to compare the precipitation volumes month by month. That precisely is what has been done in this study using meteorological data as recorded by the observatory at the University of Massachusetts, Amherst, Massachusetts.[4]

Table 1 shows the total deviation in inches of rainfall or its equivalent for the eleven-year period 1939-1949 and the preceding eleven years 1928-1938. The deviation is figured from the average for each month and year as a convenient way to decide whether the period was a wet or a dry one. The last column of figures in the table shows the total difference in precipitation between the two periods. It is most significant in that it represents an actual difference in rainfall and not just a deviation from a hypothetical "normal" period.

The fact is brought out that although the total precipitation for the years 1939-49 was 35.77 inches below average, it was 42.19 inches below the total precipitation for the preceding eleven years. When we compare the deviations in the amount of rainfall during the growing seasons (May-September) for the two periods we find that there has been 30.28 inches less during the period 1939-49 than from 1928-38. This represents 71.8 percent of the decrease in 41.7 percent

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[1] Contribution No. 780 of the Massachusetts Agricultural Experiment Station, Amherst, Massachusetts.

[2] Assistant Professor.

[3] C. I. Gunness. 1939. Meteorological Records. A fifty-year Summary. 1889-1928. Massachusetts Agricultural Experiment Station Bulletin 367.

[4] Op. cit., and Meteorological Series. Massachusetts Agricultural Experiment Station Bulletins Nos. 601-732.

Table 1. Total deviation in inches from the average precipitation during two 11-year periods, and total difference in precipitation between the two periods. Amherst, Massachusetts.

	Total deviation from 50-year average		Total difference
	1928-38	1939-49	
January	+ 5.98	- 8.55	-14.53
February	- 6.84	- 7.80	- 0.96
March	+ 1.64	+ 1.02	- 0.62
April	+ 4.07	+ 0.66	- 3.41
May	- 0.79	+ 7.12	+ 7.91
June	+11.41	+ 1.84	- 9.57
July	- 7.11	+ 2.71	+ 9.82
August	- 1.61	-12.25	-10.85
September	+16.73	-10.86	-27.59
October	- 4.73	-10.34	- 5.61
November	- 3.12	+ 5.87	+ 8.99
December	- 5.62	- 2.72	- 2.90
Annual	+ 6.42	-35.77	-42.19

of the time, an important point to note since it means that the dryness during the critical periods for growing plants has been more acute than annual averages would indicate. To be sure, the greater part of the decrease has occurred during August and September of these years, at a time when perhaps the transpiration rate is at a maximum. May, June and July have been in total above average which would tend to promote lush, tender foliage predisposed to drought injury later on in the seasons.

The amount of moisture in the soil prior to ground freeze is usually considered to be an important factor in the health of trees and shrubs. This would seem to be particularly true where evergreens are concerned since some foliar transpiration may occur throughout the winter. Precipitation on a frozen ground crust is not readily available to the roots; most of the moisture available in the soil around roots must have arrived there before the top layer of soil became frozen. For this reason rainfall during the months of August-November is particularly desirable, and a dearth of rainfall at that time is only partially compensated for by melting snow and spring rains. The records show that the total rainfall for the months of August through November 1939-1949, has been 35.06 inches less than in the corresponding period 1928-38. In other words, 83.1 percent of the total decrease has occurred in 33.3 percent of the total period, and that at a very critical time of year.

In the light of these simple facts about rainfall in Massachusetts, can we not expect that if the present cycle continues we shall suffer increasing injury to trees and shrubs, directly and indirectly a result of the weather?

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CONTROL OF CHLOROSIS IN HEATHER

R. H. Sciaroni and G. A. Zentmyer

Chlorosis or "yellows" has been known for a number of years in commercial heather grown in the field for cut flowers in San Mateo County, California. Affected plants show chlorosis, stunting, lack of new growth and, in extreme cases, severe dieback. The fungus Phytophthora cinnamomi has been found on roots and crowns of some of the chlorotic plants; there is, however, no obvious correlation between occurrence of chlorosis and heather root rot caused by this fungus.

The soil type upon which chlorosis has been principally observed is the residual decomposed granitic series common along the coastal regions of the County. Chlorosis is also a problem on alluvial overwash soils containing varying amounts of granite. In general, these soils are low in fertility and have a very low conductance. Their pH ranges from 5.5 to 6.4;[1] which would indicate that the chlorosis is not of the lime-induced type.

Growers report that in the past, soil applications of ferrous sulfate and sulfur have not given satisfactory control of the chlorosis. In July, 1949, initial experiments were conducted using soil applications of ferrous sulfate on Erica regerminans and Erica hyemalis var. Felix Faure. A solution of ferrous sulfate (commercial grade, 90% ferrous sulfate) containing one ounce per gallon of water was applied in basins around the plant at the rate of one gallon of solution per plant. After about six weeks there was indication of slight response, but greening was not pronounced in this treatment. In September, 1949, a series of spray applications was begun on E. regerminans using 0.25%, 0.5%, and 1% ferrous sulfate solutions in replicated blocks. No wetting agent or safener was added. The plants were sprayed to run-off; sprays were applied with a three-gallon knapsack sprayer. A definite response to the spray was noted in 15 to 20 days; the plants resumed growth and the normal green color was restored. The degree of greening was proportional to the concentration of the iron applied. In the untreated block the plants remained chlorotic and stunted. Because of this favorable response the grower sprayed the remaining 1 1/2 acres of E. regerminans in the fall of 1949, using a power sprayer and 175 to 200 pounds pressure. Another grower with chlorotic E. hyemalis var. Felix Faure sprayed the field with 1% ferrous sulfate just prior to cutting and obtained excellent foliage for the cut crop.

The blooms are cut from the varieties E. regerminans and E. hyemalis var. Felix Faure in November and December, and plants begin new growth again in March and April. In April, 1950, five growers started spraying fields of chlorotic E. regerminans, E. melanthera (E. canaliculata), and E. hyemalis var. Felix Faure on a five-week schedule using a 1% ferrous sulfate soltuion. About 30 acres are involved in these applications. To date the plants have regained and maintained their green color and are growing normally, while untreated areas remain chlorotic and stunted. It was noted that the response did not affect the new growth formed after the plants were sprayed; the iron is not readily translocated, a fact that has been noted by others working with different crops. For this reason repeated sprays are necessary.

Some applications have been made with a fixed-boom sprayer with two nozzles per row, others with conventional power sprayers with hand hoses. Better results are being obtained in San Mateo County with hand applications, probably as the result of better wetting and coverage. Application rates have averaged 100 to 150 gallons per acre.

This is one of the few examples of satisfactory response to ferrous sulfate sprays for control of chlorosis on a commercial scale. It is interesting to note that other crops in the same region, on the same soils, do not show chlorosis. These crops include stock, gladiolus, marguerite daisies, strawflowers and brussels sprouts.

UNIVERSITY OF CALIFORNIA, AGRICULTURAL EXTENSION SERVICE, HALF MOON BAY; AGRICULTURAL EXPERIMENT STATION, RIVERSIDE

[1]Some of the pH tests were made by Dr. D. O. Crummet, University of California, Los Angeles.

SOME DISEASES OF ORNAMENTAL PLANTS IN CALIFORNIA CAUSED BY SPECIES OF ALTERNARIA AND STEMPHYLIUM

Kenneth F. Baker and Lily H. Davis

This paper reports observations and experiments during the past decade on species of Alternaria and Stemphylium pathogenic to floricultural crops in California. All of the fungi reported produced economic damage and apparently were virulent pathogens. In addition, Alternaria raphani Groves & Skolko has recently been reported (10) as damaging to stock (Matthiola incana (L.) R. Br.) in commercial cut-flower fields in central California.

Specimens of these reported collections are available in the Herbarium, Division of Plant Pathology, University of California, Los Angeles.

Aquilegia

Mature plants of Aquilegia caerulea James were severely injured by a leafspot at Arroyo Grande, California on August 14, 1946. The spots were generally 1-2 mm. across with a light brown to dirty gray center and a dark brown margin. Only a few of these spots appeared to coalesce or become larger, but some of these reached 5 mm.; the original light center and dark margin remained even in these large spots. A Stemphylium sp. of the Eustemphylium type was sporulating on these spots. The obclavate spiny conidia had 2 major constrictions, tapered toward the apex, and were borne singly on conidiophores that showed successive swellings and which were either single or in fascicles. The disease has not been seen again, and the fungus has not been studied in detail.

Crowell and Lavalee (9) stated that Alternaria tenuissima (Fr.) Wilt. caused a leafspot of Aquilegia in Ontario, Canada. Neergaard (21) reported Alternaria tenuis auct., Stemphylium consortiale (Thuem.) Groves & Skolko, and S. botryosum Wallr. on seeds of Aquilegia caerulea and A. vulgaris L. in germinators in Denmark.

Calendula

Specimens of pot marigold (Calendula officinalis L.) with an Alternaria leaf and stem spot were received from Mr. R. H. Sciaroni, Farm Advisor of San Mateo County, California on January 10, 1949. This collection, gathered in a commercial outdoor planting in Colma, apparently is the first record of this disease in North America. The leaf spots varied in size from mere pin-points to 3/4 inch in diameter, and were irregularly elongate to circular in shape. The smaller spots frequently had distinct light colored spots in the center and rather definite brown margins. The larger lesions were brown and diffuse. On both old and young stems the lesions tended to be somewhat more elongate and to have dark margins. Similar spots occurred on the leafy bracts subtending the head, and less commonly on the petals. The disease caused considerable damage and rendered unmarketable the cut-flower crop from affected plants. It has not been found in the commercial seed fields of the State.

The fungus sporulated copiously on the leaves and on plain agar plus cold-sterilized aster stems in petri dishes outdoors. Morphologically the fungus fell within the species, Alternaria porri (Ell.) Neerg. as defined by Neergaard, and it resembled A. porri from California gerbera. However, the calendula isolate was not chromogenic, whereas that from gerbera was, as reported by Neergaard.

There are a few records of Alternaria spp. on calendula from various parts of the world but the injury does not seem to have been so great as in this case. Neergaard (21) isolated from brown poorly defined leafspots on C. officinalis in Denmark an Alternaria that corresponded morphologically to A. porri but differed in pathogenicity. Inoculation tests were successful on only 6 out of 26 calendula plants, killing 1 and severely attacking 3 others. Parallel inoculation tests with A. porri from leek and A. porri f. solani (E. & M.) Neerg. from tomato were negative on calendula. Other inoculation tests indicated that the calendula fungus was strongly pathogenic to lettuce and slightly to Godetia. Neergaard (20, No. 11-12, p. 10) reported A. porri on samples from 1 seed lot out of 47 of C. officinalis examined in Denmark in 1946, and conducted seed treatment tests (21).

Chaudhuri (7) reported an Alternaria on calendula in India which he identified as A. dianthi Stevens and Hall. Weiss (27) reported an Alternaria sp. in C. officinalis leafspots in New York, but questioned whether the fungus was causal. Alternaria tenuis was reported by Neergaard (21) as fairly common on seed samples of C. officinalis.

Callistephus

On August 16, 1946 a leafspot of China aster (Callistephus chinensis (L.) Nees.) was found in the Redondo Beach district of California producing severe localized damage in commercial cut-flower plantings. The same disease was observed in March, 1950 by Mr. R. H. Sciaroni in San Mateo and Santa Clara Counties, where the disease occurred on plants in the open and, more severely, on those under cloth. Both in central and southern California the growers involved saved their own seed and the disease probably was thus carried forward from year to year. Examination of aster fields in San Mateo County in August, 1950 revealed the disease on scattered basal leaves of most plantings, and in one location (Palo Alto) it caused severe damage higher up on the plants. The disease has not been found in commercial seed fields in the State. The causal fungus has been described (4) as Stemphylium callistephi Baker & Davis.

At least two Alternaria spp. are also known to be parasitic to China aster. Neergaard (21) reported Stemphylium botryosum on aster seeds in Denmark, and Alternaria tenuis on seed of C. chinensis from England and Hungary in 1940 and 1942. The latter was regarded as a true pathogen on aster and Godetia. Weiss (27) reported Alternaria spp., chiefly A. tenuis, as secondary in leafspots and causing seed mold in the United States. Alternaria zinniae Pape was reported on aster seeds in Denmark in 1937 and in 1942 (21); the fungus produced leaf spots on China aster, lettuce, Godetia, and zinnia. Alternaria macrospora Zimm. was reported by Hopkins (14) to defoliate large plants in wet weather in Southern Rhodesia. The large rusty leafspots could be prevented by use of a Bordeaux spray.

Dianthus

Seed flats of Dianthus caryophyllus L., Chabaud's Yellow, on March 4, 1947 at Guadalupe, California were severely attacked by a seedling leafspot and damping-off disease. The infected seedlings were about 1/2 inch high, with decayed leaves on which the fungus was sporulating copiously in dark masses. Microscopic examination of this material revealed that this disease was caused by Alternaria dianthicola Neerg. This is believed to be the first record of this pathogen or of the disease it causes in the United States. Nearby flats of Early Dwarf Vienna Mixed and J. M. Bridgeford had only slight infections. The evidence suggested that the fungus was seed-borne in the lot of Chabaud's Yellow, and this was verified by planting some of the same lot of seed in pasteurized soil in the University greenhouses at Los Angeles. These seedlings began to die off shortly after emergence and there was evidence of a high percentage of transmission with the seed. On investigation it was found that the Vienna and Bridgeford varieties were grown in Guadalupe in 1945, whereas the Chabaud's Yellow was grown there in 1946. The record showed that plants of the latter variety had been exposed to at least one rain of 0.13 inches on October 16 (and perhaps another of 0.34 inches on October 1) while piled on canvas sheets in the fields prior to threshing. It is more than probable that the moist conditions resulting from the rain greatly favored the spread of the fungus into the seed.

The fungus was observed by us again in May, 1947 in light-colored spots on the petals of a carnation flower purchased in a Los Angeles florist's shop.

The fungus had previously been found by Neergaard (letter of May 22, 1947) in Ottawa and in the Montreal Botanic Garden on Dianthus caryophyllus in August, 1946. This was reported by Savile (27) and probably constitutes the first record of this fungus in North America.

Since A. dianthicola was described by Neergaard in 1942 (20, No. 7, pp. 8-9; 21) it has been found to be the most common Alternaria pathogenic on Dianthus in Denmark. He (20, 21) found the fungus in 38.5 percent of the 192 samples examined from 1941 through 1944 and in 32.8 percent of 262 carnation seed samples examined from 1945 to 1947. Eradication of the fungus from carnation seed was not accomplished by a 5-hour immersion in 0.5 percent Germisan (21). In the period 1941-44, 13.0 percent of 23 seed lots of D. barbatus L. were also infected with A. dianthicola (21). It is quite possible that the fungus was introduced into North America by seed.

Alternaria dianthicola has been reported occurring spontaneously on D. caryophyllus, D. barbatus, and D. plumarius L. in Denmark, and was successfully inoculated on Gypsophila elegans Bieb. In Denmark the fungus is said to cause light colored spots with dark margins on leaves, stems, and buds, but apparently is not incriminated in that area in the production of a branch rot symptom. It is not known whether A. dianthicola will produce branch rot in the United States. The description of the fungus that attacked carnation and Sweet William in Italy in 1937-38 (8) suggests that it may have been A. dianthicola.

Alternaria dianthi produces a characteristic branch rot symptom, as well as leafspots, in outdoor plantings and to a lesser extent in glasshouses in California. Bickerton (5) reported

A. dianthi on Dianthus caryophyllus, D. tristis Vel., D. barbatus, D. plumarius, D. chinensis L. var. heddewigii Regel, and D. allwoodii Hort. The fungus was also reported by Weiss (27) as questionable on D. deltoides L. and as generally distributed over the United States. The possibility exists that A. dianthicola is being reported under the name A. dianthi in the same way that it was in Denmark prior to their differentiation.

Gerbera

Seedlings of Transvaal daisy (Gerbera jamesoni Bolus) about 1/2 inch high were received in July, 1948 from Guadalupe, California. They had numerous leafspots about 1 mm. in diameter, with light tan centers and darker margins. Eventually these spots spread until the leaves were killed, and the seedlings sustained serious injury. In September, 1948 leaves of mature plants were received from Pismo Beach, California. These leaves had many spots ranging from 1 to 8 mm. in diamteer. The small spots were dark reddish brown and frequently had a tiny white area in the center. As the spots enlarged, the darkened areas remained and a light colored zone developed around them. Alternaria porri was present on the larger spots. Additional material recieved in December, 1948 from the same place had similar but more numerous leafspots. The same leafspot was again collected in September, 1949 on mature plants at Oceanside, California by Dr. J. G. Bald. While most of the leafspots in this collection were small, some had attained a diameter of 1 cm., and tended to have reddish margins and brown centers with a small white spot in each.

What appears to be the same leafspot was described in Italy by Elisei in 1938 (12). He identified this fungus as Alternaria solani (E. & M.) Sor., but, as was pointed out by Neergaard (21), he conducted no inoculation tests on either Gerbera or on potato, and this fungus may, therefore, have been any form of Alternaria porri. Hopkins (14) listed a brown leafspot and flower blight of Gerbera as widespread in Southern Rhodesia. He considered that this was caused by Alternaria macrospora, which Neergaard (21)places in A. porri. The leafspot and rot of the petals and flower heads was said to be controlled by the use of a Bordeaux spray.

Alternaria zinniae was reported (20, No. 11-12, p. 11) on 1 out of 8 seed samples of Gerbera examined during 1946. Alternaria tenuis likewise occurs on the seeds of Gerbera (21). While it has not been definitely established that Alternaria porri is seed-borne on Gerbera seed in California, the observational evidence indicates that such is the case. The seed beds where the disease was first observed were not near mature infected plants, and the development of the disease on the seedlings was indicative of a local source of inoculum.

The use of a Bordeaux spray on the mature plants of Gerbera in the field in coastal California has given fairly effective control of this leafspot.

The Alternaria leafspot is very similar to the one caused by Ascochyta gerberae Maffei that is even more common on cultivated Transvaal daisy in California. The Ascochyta leafspot was first observed in May, 1941 but was undoubtedly present before that time as it was then widespread from San Diego County to the San Francisco Bay area. Another leafspot which might be confused with that caused by Alternaria was collected near Orlando, Florida on June 15, 1948 by the senior author on outdoor-grown mature plants. The fungus in this instance was identified by Dr. Charles Chupp as Cercospora gerberae Chupp and Viégas and was said (letter of August 16, 1948) to be a new record for the United States.

Gladiolus

The red leafspot or Stemphylium leaf blight of gladiolus is an important disease in commercail cut-flower plantings in California. Apparently this disease was first observed in California by Dr. J. T. Barrett in 1937 at Santa Maria on Picardy and one other variety, but this was not confirmed by culturing. He observed it again in April, 1939 in San Mateo County and on specimens submitted from San Diego County. The disease was particularly severe on the Picardy variety but occurred on a few others. It produced reddish brown, slightly depressed spots 1/2 to 4 mm. in diameter or sometimes larger if the spots coalesced. A Pleospora sp. was isolated from 50 to 75 percent of the spots. It was observed by the senior author in February, 1940 in South San Francisco, and in May at Irvington and on specimens sent in from Eureka. It was observed again in July, 1941 at Oceanside, California where serious injury was produced on Picardy, Salbach's Orchid, and Minuet varieties, but the adjacent variety, Rapture, was only slightly injured. Again a Pleospora sp. was isolated from the leafspots.

The characteristic symptom of the disease appears to be the tiny red or brown spot in the center of the yellow, translucent, round spots that are up to 1/8 inch in diameter (22). The

disease has been reported (22) in Michigan, California, Florida, New York, and New Jersey, and apparently was observed in Florida as early as 1940.

This leafspot is favored by moist cool conditions. Because infection can occur with as little as 10 hours of continuous moisture and is favored by average temperatures of 55° to 75° F. (18), the severity of the disease in fields watered with overhead sprinklers in the coastal fog belt of California is to be expected.

The degree of susceptibility differs markedly with different varieties. A tabulation of these was presented by Magie (17). He (16) has obtained effective field control of the disease in Florida by using Dithane-zinc sulfate-lime, Zerlate, or Parzate sprays applied once a week or oftener during wet weather.

Nelson and Mooar (23) obtained a Pleospora stage of the fungus in a single culture, but did not identify the species. The California Stemphylium is similar to S. botryosum Wallr. sensu Wiltshire, and the perfect stage which is commonly produced by it in culture resembles Pleospora herbarum (Pers.) Rabenh. Alternaria fasciculata (Cke. & Ell.) Jones & Grout has been reported on gladiolus in Pennsylvania (1). Macrosporium commune Rabenh. has also been reported in Southern Rhodesia, an Alternaria sp. in Holland (19), and a Stemphylium sp. in Italy (7a).

Lunaria

A leafspot was observed in a commercial seed field of honesty (Lunaria annua L.) at Lompoc, California on May 1, 1941 following a very wet winter. There were abundant spots about 1 mm. in diameter scattered over the leaves, particularly on those at the base of the plants. Many of these spots had a distinct dirty gray central dot surrounded by a broad dark brown rim; some superficially gave the appearance of bacterial lesions. A small number of these spots continued to enlarge and, although the original dark spot remained, a light tan circular area, in many cases without any distinct margin, developed surrounding it. As these enlarged and coalesced they frequently became highly irregular in shape, and 2-4 cm. across. The causal fungus was sporulating sparsely on all but the very smallest leafspots. A few slender, linear, brown spots 1 mm. long were observed on some of the petioles.

Microscopic examination of the fungus showed that it was well within the limits of Alternaria brassicae (Berk.) Bolle as defined by Neergaard (21). While this fungus has apparently not heretofore been reported on Lunaria, it occurs on a wide range of other cruciferous plants.

Weiss (27) reported an Alternaria sp., possibly A. oleracea Milbrath (= A. circinans (Berk. & Curt.) Bolle) that caused a black spot of leaves and pods of Lunaria in Massachusetts.

Saponaria

A severe outbreak of a leafspot disease was observed in a small seed planting of Saponaria vaccaria L. in Lompoc, California on April 30, 1940. The leafspots were at first less than 1 mm. across, were deep brown, and sometimes had a tiny white fleck in the center. These leafspots enlarged and ordinarily became about 3 mm. in diameter although in some cases they attained 7 mm.; the original dark area of the small spot remained and the advancing lesion beyond this area became tawny or dirty gray, and was covered with dark sporulation of the fungus. A narrow dark brown band continued at the advancing margin of the spot. On the stems the spots were linear, attaining a length of 3-4 mm., with white centers and dark margins. At the base of the older stems the fungus advanced down from the leaf axils, producing extensive lesions surrounding the stem and sometimes extending downward for as much as 2 cm.; on such lesions there was copious sporulation. The general picture of this type of infection was similar to that of carnation branch rot. Spots were produced on the calyx of the flower heads and it is probable that the fungus spread to the seeds.

The fungus, which was collected again at the same place in April and June 1942, caused extensive damage to the seed planting. It was present but caused less damage in June, 1945.

Examination of these specimens showed that the fungus corresponded to Alternaria saponariae (Pk.) Neerg. This fungus is reported to cause leaf and stem spot of Saponaria officinalis L. from Connecticut to Maryland and in Indiana and Minnesota (27). Neergaard (21) considers A. saponariae (Macrosporium saponariae Pk.) to be identical with A. dianthi, and our study of these specimens supports this view. Macrosporium saponariae was collected by Peck on leaves of Saponaria officinalis in Alcove, New York in September, 1893, but there must have been a previous collection since the species was described from Greenbush, New York by Saccardo (Vol. 4) in 1886. The same fungus was collected by Rostrup on S. officinalis in Denmark in 1897 and was seen there on S. vaccaria in 1937 by Neergaard. Macrosporium saponariae was

reported on Saponaria officinalis in Manitoba (9) and Esthonia (15), and A. dianthi was found on the same host at Ankara, Turkey (6).

Senecio

An attack on cineraria (Senecio cruentus DC.) by Alternaria senecionis Neerg. was observed in the University greenhouses, West Los Angeles on November 10, 1942. The fungus caused damping-off and leafspot of seedling cinerarias growing in a flat of steamed soil under conditions that strongly suggested seed transmission. Mature cineraria plants with conspicuous leafspots were observed in Los Angeles County in January, 1943 and January, 1947. The fungus isolated from the leaves was typical of Alternaria senecionis.

Inoculation tests[1] were conducted in February and March, 1947 using suspensions of spores and spores plus mycelium. The plants were held for 48 hours in a moist container. Within 7 to 10 days leafspots developed on many of the leaves, whereas the checks remained normal. The spots were generally very irregular in shape and size; when they occurred on or near veins they tended to elongate and extend for a distance up to 1 cm. Some of the large leafspots became 2 cm. across with highly irregular margins. Generally the spots were brown in color, the large ones lacked a border but the smaller ones had dirty gray to whitish central areas and dark brown margins. Petiole lesions were frequent, linear, and up to 2 mm. long. Profuse scattered infections resulted in every inoculation test. Inoculations of zinnia were unsuccessful. Parallel inoculations with A. zinniae were successful on zinnia but not on cineraria.

Neergaard (in a letter dated May 22, 1947) stated that ours apparently was the first record for this fungus on cineraria outside of Denmark. He found it for the first time in 1943 on Danish, French, and German seeds in a germinator in Denmark (21). His inoculation tests showed that the fungus was able to infect cineraria and lettuce strongly, and tomato, cucumber, and Godetia slightly. The disease was fairly common in Denmark and has also been reported (13) in England and Ireland. Because the fungus was considered to be seed-borne, experiments were conducted by Neergaard (21) to determine the efficacy of seed treatment. A 1-2 hour soak in 0.5 percent Germisan freed the seeds of A. senecionis and A. tenuis without host injury.

Alternaria tenuis has been reported (21) on seeds of Senecio cruentus. Alternaria zinniae was also found in 1943 on seeds of cineraria in a germinator in Denmark; this is the first report of the fungus on this host (20, No. 9, p. 8; 21).

Zinnia

Apparently the first collection of Alternaria zinniae Pape in California was made by the senior author on August 1, 1941 in a commercial seed field of zinnia (Zinnia elegans Jacq.) in Lompoc. Leafspots were few, about 1 mm. across, with typical light centers and dark margins, and were so inconspicuous that they were detected only because a specific search for the disease was under way. The same fungus was found on a few leaves in a large commercial seed planting in El Monte in September, 1942. Evidences of the disease have since been observed infrequently in California seed fields; leafspots are extremely rare, stem lesions have not been seen, and in only one instance (on variety Will Rogers) was any evidence of possible root injury from the fungus observed. In 1942 and again in 1943 the fungus was found causing conspicuous leafspots on zinnia seedlings in flats in 2 Los Angeles nurseries. Since these plants were grown under shaded conditions and watered from above, this outbreak of symptoms, typical of those that normally appear in the eastern rainy districts, was to be expected. These two collections most nearly resembled the eastern leafspot phase of any made in California. Symptoms of the trouble as they appear in the eastern States have been described by Dimock and Osborne (11).

It is probable that this disease has been present in the United States for a long time, possibly since 1924, when an Alternaria leafspot of zinnia was collected at Lake Alfred, Florida by G. F. Weber. The disease was of such importance in New York by 1934 that studies were begun at Cornell University. In the United States it is also reported (27) in Connecticut, New Jersey, New York, Pennsylvania, and South Carolina. The first known record of the fungus was on July 22, 1902 in Copenhagen, Denmark when it was observed by Rostrup (21). It is by now apparently widely distributed over the world, occurring in Canada (British Columbia and Ontario), Denmark, England, Germany, Holland, France, Hungary, Italy, and Southern Rhodesia. Neergaard reported A. zinniae on 71.4 percent of 448 zinnia seed samples studied in 1939-1943, and in 43.8 percent of 178 samples examined in 1945-47.

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[1]The assistance of Dr. A. W. Dimock in some of these tests is gratefully acknowledged.

Alternaria sonchi J. J. Davis is reported (26) from Brazil on Z. elegans. This fungus does not appear to be A. zinniae.

Alternaria zinniae apparently infects a number of plants, including tomato, lettuce, Godetia, Nicotiana sanderae Sander, N. alata var. grandiflora Comes (= N. affinis), Zinnia elegans, Zinnia haageana Regel, and China aster (21).

Alternaria zinniae is known to live for 5 years in zinnia seed (21). Internal seed transmission has been verified by Pape (24) and by Dimock and Osborne (11). Because most of the zinnia seed of the world is grown in California, it seemed possible that the infected seed which was reported in other areas may have originated in that State. Therefore, beginning in 1941, extensive field surveys were made to determine whether the disease was present in the seed fields.

The fungus only rarely produces symptoms here of the type reported for other areas. When examined in the fall of 1944, following an exceptionally cool and humid summer, no recognizable symptoms were detectable on plants in a field known to have been consecutively planted to zinnias for 6 years. The same absence of symptoms had been observed there in 1943. Seed from the field in each of these years was found, however, to be infected when indexed in the greenhouse. The disease probably was present and the fungus disseminated on California seeds for some years before it was discovered.

The sequence of the disease in a California seed field is now considered to be as follows: The primary inoculum is from (a) infected seed that is drilled directly into the fields, and (b) soil carryover, the usual 2- to 3-year rotation being insufficient to eliminate the fungus (11). Because of the semi-arid climate it is probable that only an occasional seedling actually dies or develops typical cotyledonary lesions, although under the cloudy or foggy conditions prevalent along the coast this may occur when irrigation is practiced. However, as the plants get larger the dry conditions essentially eliminate leafspots, stem lesions, or even typical petal spots. The fungus during this period probably persists in a few tiny lesions on leaves or on dead seed lings on the soil surface. By mid-summer plants in the field have lesions from many other causes (e.g., insect feeding, or cultivation, wind, and spray injury), so that a few Alternaria spots would easily be overlooked. Observations by the senior writer in New York further indicated that considerable flower damage can result even when there are relatively few leafspots.

In the late fall, when zinnia plants in California are becoming senescent, most of the flower heads have dried and shrivelled. During this season in the coastal seed fields, night fogs and heavy dews are not uncommon. It has been noticed repeatedly that flower heads become saturated with moisture and remain wet through much of the following day, whereas the leaves either remain dry or become so in less than an hour after the sun comes out. Air-borne spores of A. zinniae that fall on moist flower heads would infect, and the fungus then rapidly and copiously sporulate. The number of infected senescent and dead petals could therefore increase quickly from an initial low inoculum. Numerous other saprophytic fungi of the genera Alternaria, Cladosporium, Botrytis, etc., also develop on these flower heads. For this reason it is practically impossible to determine by field inspection whether or not the seed produced is carrying Alternaria zinniae. This Alternaria undoubtedly decays the petal and progresses downward directly to the attached seed and grows into the fruit coat. In addition, spores of the fungus could be carried in threshing to many of the other seeds as well.

There are two general methods of harvesting zinnia seed in California. In one the seed heads are hand-picked as they mature and are piled on canvas on the soil to dry before threshing. In the other, the whole plant is cut and is put through a threshing machine or is "combine-harvested" in place. In either case it is the general practice to wait until the first frosts before threshing, as those tend to loosen the seeds from the "cone" (receptacle) and make cleaning easier. This means that the seed heads usually remain in the field after the season of fog and heavy dew in the coastal area. Infection of zinnia occurs readily at 65° F. (11) and it is probable that it can occur at lower temperatures.

This set of circumstances explains how a fungus that rarely produces a recognizable disease of the California seed crop may infect the seed and thus be carried to new areas. In only one other instance in California-grown flower seed has a pathogen been found to contaminate seed when no evident disease was produced in the field. This was in the case of Corynebacterium fascians (Tilf.) Dowson on nasturtium and sweetpea (3).

Control

Seed Treatment Tests: --

Pape (24) was able to disinfest zinnia seeds by immersing them for 1/2 hour in 0.125 percent Germisan or Uspulun. Neergaard (20, No. 5, pp. 9-11), on the other hand, was unable to

disinfest the seed even in 3 hours at the same strength of Germisan or at 0.25 percent Uspulun, and the germination of the seed was retarded by the treatment. This discrepancy probably arose from internal transmission in the second instance but not in the first.

Because of internal transmission of A. zinniae, no chemical treatments of the seed were attempted. Preliminary tests were conducted to determine the tolerance of zinnia seed to hot water. Treated and untreated seeds of the 1943 crop of Dahlia Flowered Polar Bear, Dahlia Flowered Golden State, and California Giant Enchantress were sowed in flats of pasteurized soil and kept very moist in a greenhouse. Data were taken (Table 1) after 8 and 21-24 days. Only the variety Polar Bear was infested by A. zinniae, and in this lot the seedling stand decreased 48.3 percent in 13 days owing to attack by the fungus, whereas the stand of the other varieties increased during that time. Enchantress sustained a 32.3 percent reduction of stand from the 51.5° C. treatment, and Golden State 4.4 percent; the stand of Polar Bear was reduced about 19.5 percent, but the fungus was killed. As a result of this and other tests, as well as treatment of many hundreds of pounds of commercial zinnia seed, the use of hot water at 51.5°C. (125° F.) for 30 minutes has been adopted as standard against this pathogen.

Table 1. Effect of hot-water treatment on germination of new seed of three zinnia varieties. Planted January 26, 1944 in pasteurized soil.

Seed treatment in hot water for 30 min. at indicated temperature	Date	Percentage seedling stand		
		Polar Bear[a]	Golden State	Enchantress
Untreated check	2-3-44	87	63	63
	2-16-44	45	90	96
49° C.	2-3-44	73	67	63
	2-19-44	83	92	84
51.5° C.	2-3-44	64	75	53
	2-19-44	70	86	65
54.5° C.	2-3-44	52	41	40
	2-19-44	59	52	47

[a]Seed infested by Alternaria zinniae; other varieties uninfested.

This treatment does not seriously reduce the germination of new seed, but frequently is injurious to that 2 years or more old. This is shown by the results (Table 2) of treating 12 seed lots of varying age. Samples of treated and untreated seeds of each variety were planted in flats of pasteurized soil. No Alternaria developed on any of these seed lots. Data were taken on seedling emergence after 65 days. First-year seed of 7 varieties had an average of 15.6 percent reduction of stand, second-year seed of 4 varieties averaged 38.9 and one third-year lot had a 78.8 percent reduction.

The heat treatment is primarily suggested for seed to be used in planting fields for commercial seed production, and in such use the germination reduction from the treatment is unimportant. It is also unlikely to be noticed in garden plantings, but it may attain significance when seed is sown in flats for bedding-plant sale. Because of the large volume of zinnia seed sold each year, treatment of seed before marketing is impractical. Treatment of basic seed stocks that are planted in proper climatic areas is regarded as the best means of producing uninfested seed for retail sale.

Cultural Practices: --

Selection of the seed-growing area has become an accepted method for commercial production of disease-free seed (e.g., cabbage and cauliflower against black rot and black leg, beans against anthracnose and bacterial blights). The possibility was investigated that such a method might be found for Alternaria blight of zinnia. Much of the seed has in the past been grown in the coastal fog belt of California or in areas having heavy dew or fog prior to seed harvest in the fall.

Table 2. Effect of hot-water treatment on germination of zinnia seed of varying ages. Planted in pasteurized soil on April 5, 1945. Data taken June 9, 1945. Alternaria zinniae not present in these lots.

Variety	Percentage germination in soil	
	Untreated	Treated
1944 SEED CROP		
Dahlia Flowered Scarlet Flame	56.7	41.6
" " Mixed	66.6	51.6
California Giant Scarlet Queen	64.6	49.5
" " Enchantress	70.5	55.6
Crown o' Gold	59.0	54.1
Zinnia haageana	79.0	76.0
Fantasy Mixed	62.7	59.1
Averages, 1944 seed	65.6	55.4
Average germination reduction of 15.6%		
1943 SEED CROP		
California Giant Daffodil	70.3	45.0
Lilliput Mixed	63.6	46.0
Howard Crested	73.5	20.0
Gracillima Red Riding Hood	56.6	50.1
Averages, 1943 seed	66.0	40.3
Average germination reduction of 38.9%		
1942 SEED CROP		
California Giant Lavender Gem	37.8	8.0
Germination reduction of 78.8%		

Zinnia seed obtained from the 1943 and 1944 crops from various coastal and interior areas of California was indexed in the greenhouse for A. zinniae, with the following results:[2]

Coastal California:

Buellton -- Dahlia Flowered Golden State, A++3; California Giant Enchantress, A++3.
Arroyo Grande - California Giant Choice Mixed, A++3.
Saticoy -- Dahlia Flowered Polar Bear, A++R3.

Inner-coastal California:

Santa Paula -- California Giant Scarlet King, 03; Super Giants Mixed, 04; Dahlia Flowered Canary Bird, 04; D. F. Royal Purple, 04; D. F. Exquisite, 04; D. F. Golden Dawn, A+4.
El Monte -- Dahlia Flowered Polar Bear, A+++34; D. F. Canary Bird, 04; D. F. Dream, A+4; D. F. Crimson Monarch, 04; D. F. Golden Dawn, 04; D. F. El Dorado, 04; D. F. Golden State, 0R4; California Giant Purity, A++4; C. G. Enchantress, 04; C. G. Brightness, 04; Double Giant Scarlet, 0R4.
Fillmore -- Dahlia Flowered Crimson Monarch, 0R3.

Interior California:

Delano -- Dahlia Flowered Polar Bear, 0R3; D. F. Scarlet Flame, 0R4; D. F. Golden State, 04; D. F. Exquisite Mixed, 04; D. F. Crimson Giant, 0R4; D. F. Purple Prince, 04; D. F. Canary Bird, 04.

[2] A = Alternaria zinniae present in amount indicated by number of + signs. 0 = A. zinniae absent. R = Rhizoctonia solani present on seed, otherwise absent. 3 = 1943 seed. 4 = 1944 seed

Rocky Ford, Colorado:
California Giant Purity, A++3; C. G. Miss Willmott, 03; Dahlia Flowered Polar Bear, 03; D. F. Exquisite, 03. This seed from a Colorado seedsman probably was grown in that State. Because growers sometimes market another's seed in their own packets, this case can be only presumptive.
Doylestown, Pennsylvania:
Fantasy, A+++4; Hybrid seed of 3 populations, A++4; A++4; and A+++4.

From the above data it is evident that seed from coastal and inner-coastal fields in California is commonly infected with Alternaria, whereas the probability exists that seed produced in dry interior valleys of the State may be free of the disease. Because the plant appears to develop and seed well in these hot dry valleys, growers should further explore the possibility of producing zinnia seed in the San Joaquin, Coachella, Antelope, Hemet, and Imperial Valleys. This is especially desirable because the other principal zinnia seed-producing areas of the country (Colorado, New York) apparently produce Alternaria-infected seed.

Rhizoctonia solani occurred in 7 seed lots from Saticoy, El Monte, Fillmore, and Delano, whereas Alternaria did not occur in the latter. This suggests that Rhizoctonia invades zinnia seed (2) in a different manner than does A. zinniae.

Harvesting and threshing practices are important in determining the degree of infestation of the seed by A. zinniae. These operations should be completed before the fall period of fog and heavy dew, particularly along the coast of California but probably also in the interior valleys. This may require development of a type of threshing equipment better able to separate seed from the fragments of broken receptacle.

The seven 1944 seed lots in Table 2 were not contaminated with either A. zinniae or R. solani, despite the fact that the land on which they were grown was known to be heavily infested and to have been planted to zinnias for 6 consecutive years. Furthermore, seed from the 1943 crop in this field had been indexed and found to be infected. The summer of 1944 was unusually cool, foggy, and favorable for Alternaria as evidenced by the heavily infected seed lots reported in this paper. The seven lots in question had been machine-threshed from standing plants, and the flower heads thus were not piled on canvas on the ground in the usual way. It is possible, therefore, that the anomalous freedom from infection in these lots may have resulted from the method of harvesting.

Indexing of Seed Lots: --

Because of difficulty in determining by field examination in California whether the seed produced is infected by A. zinniae, an indexing method was devised. Seed is sowed thickly (about 1000 per 18 x 18 in. flat) in soil that has been pasteurized in the flats, and is thinly covered with additional pasteurized soil. The soil is kept very wet to favor development of any Alternaria present and data are taken in about 20 days. Black lesions girdle the stems of the seedlings at soil level and may extend upward for an inch or more; on these lesions Alternaria may sometimes sporulate. The cotyledons frequently exhibit infections that appear to originate from the seed coat which remains attached to them for some time. The basal leaves may develop typical leafspots if they are kept wet enough. This method gives a reliable means of determining seed lots that are infected and require treatment, but may give an exaggerated picture of the amount of infection unless data are taken before secondary spread occurs.

Summary

Several species of Alternaria and Stemphylium have been observed causing diseases of ornamentals in California during the past decade. These are listed below, and the year indicated when each was first observed in the State:
Alternaria brassicae: Lunaria annua, 1941; apparently a new host record.
Alternaria dianthi: Dianthus caryophyllus, long present in State. Saponaria vaccaria, 1940.
Alternaria dianthicola: Dianthus caryophyllus, 1947; probably first U. S. record of fungus.
Alternaria porri: Calendula officinalis, 1949; apparently first record in U. S. on this host.
Gerbera jamesoni, 1948; apparently first U. S. record on this host.
Alternaria raphani: Matthiola incana, 1946, apparently first U. S. record on this host (10).
Alternaria senecionis: Senecio cruentus, 1942; apparently first record for fungus in North America.
Alternaria zinniae: Zinnia elegans, Z. haageana, 1941.
Stemphylium callistephi: Callistephus chinensis, 1946; first record of fungus (4).
Stemphylium (botryosum ?): Gladiolus spp., 1939.

Alternaria zinniae was found to commonly infect California-grown zinnia seed, even though symptoms of the disease were so rarely observed in the field as to render surveys useless. Indexing of seed by planting in moist flats of pasteurized soil was necessary in order to determine whether given lots were infested and required treatment.

Because A. zinniae is internally seed-borne, a hot-water treatment was devised for infested lots used for planting commercial seed fields. Seed is immersed in water at 51.5° C. (125° F.) for 30 minutes, cooled rapidly, and dried. Germination reduction from the treatment is not serious for one-year-old seeds, but may be significant with older lots.

The possibility of avoiding seed infection by selection of the growing area is sufficiently promising to merit consideration by producers. Because infection in California probably occurs in the fall following the advent of night fog and dew, seed harvest should be completed prior to that time. Certain indicated areas of the State have a minimum of fog, and production of the crop in these localities by planting treated seed in time to permit early harvesting should almost eliminate infested seed from the market.

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UNUSUAL RECORDS OF PLANT DISEASES

THE REAPPEARANCE OF SILVER LEAF IN THE APPLE ORCHARDS OF WASHINGTON STATE[1]

By Roderick Sprague and H. H. V. Hord

In the autumn of 1923 silver leaf was found to be prevalent in apple orchards in the Spokane Valley and some other parts of eastern Washington. Investigation by Heald and Boyle[2] established the fact that this disease had been recognized as causing loss in the upper Columbia country as early as 1916-1917. A thorough study of the problem by Heald and his students, carried on from 1923-1926, correlated the activity of the causal organism Stereum purpureum with winter injury of the trees. Infection by S. purpureum was found to be almost invariably followed by the death of the branch or, if allowed to run its course, by the death of the entire tree. Silvered branches died either the same season or after one to several seasons. Previous to the death of the infected parts, a general decline in vigor was observed either in particular limbs or in the entire tree. When the affected parts were dead, the fruiting bodies of the fungus began to make their appearance upon the surface of the bark.

Silver leaf symptoms were again observed this autumn for the first time in a number of years Severe symptoms were observed on the varieties Red and Golden Delicious, Johnathan, and Rome Beauty, though these were previously considered to be less severely affected than the Wagener variety.

Symptoms were found in localized areas in the Entiat, Methow, Okanogan and Yakima Valleys where trees had suffered considerable damage from the severity of the 1948-1949 and 1949-1950 winters. Mature trees that had been partially winter killed commonly showed the silver leaf symptoms but young trees without any obvious winter killing were also found affected. In one small five-acre orchard in the Okanogan Valley, consisting of trees five to six years old, more than 90 percent of the trees were found affected. Newly developed sporophores of Stereum purpureum were observed on dead bark at the base of two affected trees on October 7 following the early fall rains.

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ARTICULARIA ON PECAN IN TEXAS

By John R. Cole

Recently Mr. Louis Romberg, in charge of the Brownwood, Texas laboratory, made a collection of Articularia quercina var. minor on pecan at the Brownwood station. This fungus was first collected on pecans by Mr. Demaree and myself along the Colorado River at Utley, Texas, in 1933. Mr. Demaree forwarded this material to Miss Vera K. Charles and she described the fungus in the January-February 1935 issue of Mycologia. According to Miss Charles there has only been one previous collection of this fungus and that was made by Dr. L. E. Miles in 1920 at DeSoto, Mississippi on leaves of Quercus sp.

U. S. DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, PECAN FIELD STATION, ALBANY, GEORGIA.

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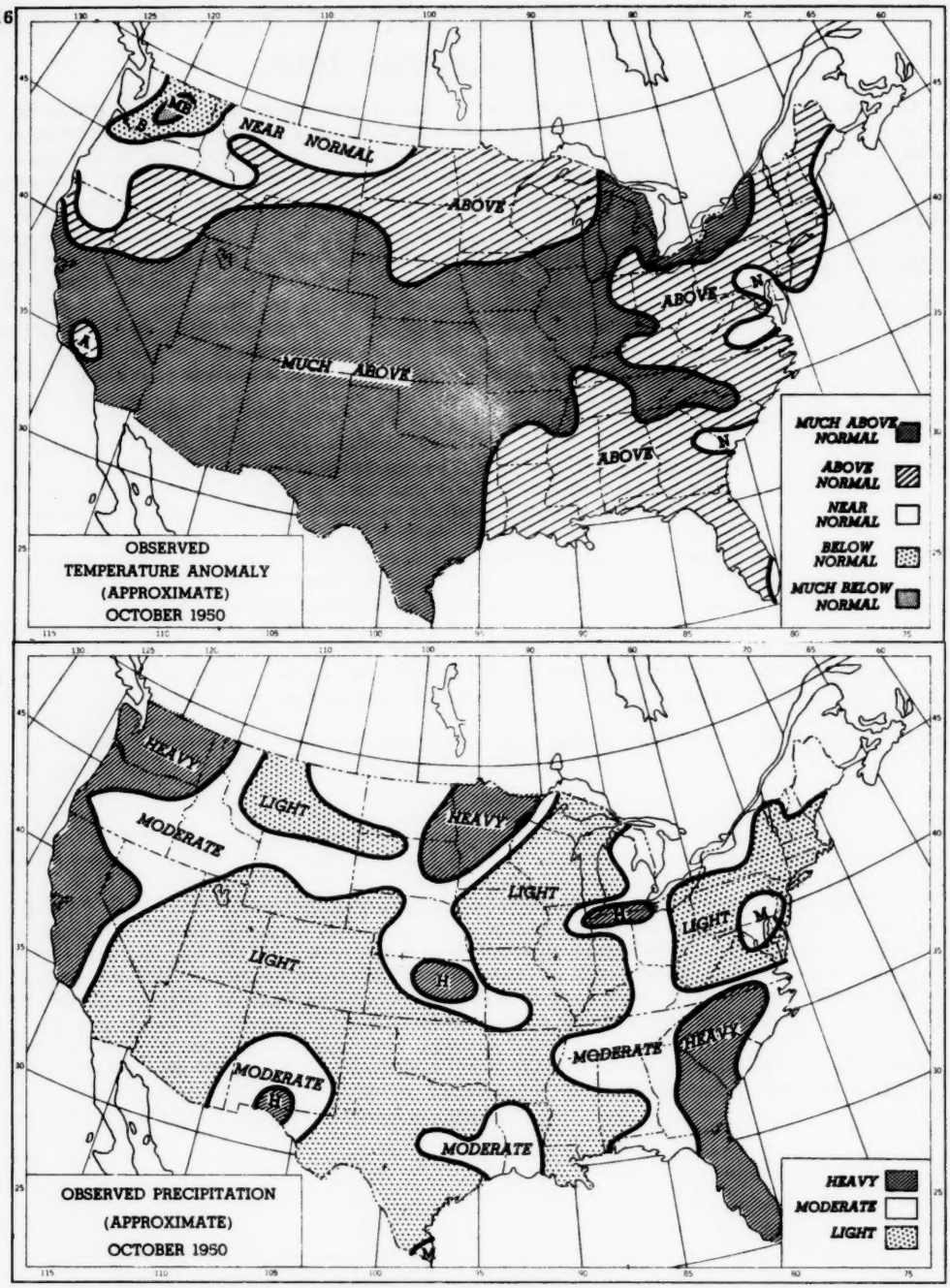
[1] Published as Scientific Paper No. 970, Washington Agricultural Experiment Stations, Institute of Agricultural Sciences, State College of Washington, Pullman, Washington.

[2] F. D. Heald and L. W. Boyle. The Menace of Silver Leaf, Proceedings of Washington State Horticultural Association. 19: 50-54, 1923.

CORRECTIONS FOR SUPPLEMENT 192

On page 167 of Supplement 192, "Nation-wide Results with Fungicides in 1949" it was reported that Robertson's Copper seemed to be ineffective against early and late blights of tomatoes and early blight of potatoes. It has been called to our attention that Robertson's Copper was as effective in controlling late blight of potatoes as the fixed coppers although both were less effective than the organic sulfurs. In limited tests Robertson's Copper looked promising for the control of early and late blights of tomato.

On page 178, paragraph 3, line 7, should read, "With both, the least effect ....." instead of maximum effect as given.



The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.